

RESPIRATORY CARE

A MONTHLY SCIENCE JOURNAL
40TH YEAR—ESTABLISHED 1956

SPECIAL ISSUE
Resuscitation in Acute Care Hospitals
Part II

- Ventilation During CPR
- Emergency Drug Administration
- When is Enough, Enough?
- DNR Orders: A Critical Review
- ACLS Skills & Knowledge Retention
- Maintaining ACLS Skills in Hospitals
- ACLS Training for Students
- Neonatal & Pediatric Resuscitation



41st Annual Convention and Exhibition
December 2-5 • Orlando, Florida

Last Call for 1995 OPEN FORUM Abstracts

Sooner or later, you'll be weaning him off the vent.



So why not make it sooner?

Weaning protocols that use capnography can help take the guesswork out of weaning decisions.

Considering the time, resources, and quality-of-care issues involved in weaning ventilated patients, the implications are clear. When the process of weaning patients from the ventilator is arbitrary, it creates variability that can lead to increased costs and reduced efficiency.

There is an alternative. Close, continuous monitoring of end-tidal CO_2 —as part of a weaning protocol—provides



With the ULTRA CAP monitor, the effects of ventilator settings can be measured breath to breath, rather than after the 10- to 20-minute waits associated with blood gas analysis.

timely information to help you gauge your patient's ability to be weaned off the ventilator. Instituting a protocol that leads to fewer ABGs and reduced ventilator time per patient can save money, potentially improve quality of care, help reduce MLOS, and make bed utilization more efficient.

Make the ULTRA CAP monitor a part of your ventilator weaning process.

Nellcor can help you integrate capnography into a vital and effective weaning protocol. Take advantage of our extensive training and support materials, including our comprehensive collection of institutional weaning protocols. And you can use the ULTRA CAP™ monitor—Nellcor's premier capnograph and pulse oximeter—to implement a protocol of your own.

Because when it comes to weaning patients off the ventilator, we think you'll agree—the sooner, the better.

For more information, contact your local representative or call 1-800-NELLCOR or 510-463-4000. (Call our European office at +31.73.426565 or our Asia/Pacific office at +852.529.0363.)

NELLCOR®

NELLCOR and ULTRA CAP are trademarks of Nellcor Incorporated.
© Nellcor Incorporated 1994. All rights reserved.

Circle 109 on reader service card

Summer Forum Is Approaching



Start planning now to bring the whole family
and join your RC colleagues in beautiful
Vail, CO, July 14–16. Look for registration
and program information in *AARC Times*.



See you there!



"When it comes to versatility and aerosol therapy, no other MDI spacer delivers like ACE®."

Thanks, DHD."

*Joe Nicoletti, RRT,
Coordinator of
Respiratory Therapy*



With the ACE Aerosol Cloud Enhancer, you're assured of fast,

effective MDI delivery in every situation. In addition to routine, oral inhalation, ACE's versatile design permits use in a vent circuit, in conjunction with an endotracheal airway or resuscitation bag, and with an incentive spirometer. Its patented, cone-shaped chamber maximizes respirable volume, while its clarity allows you — and your patients — to easily confirm availability of prescribed dose. For more information on ACE, the ACE Patient Training Video, or the entire line of DHD quality respiratory care products that RTs appreciate, please call DHD toll-free at

1-800-847-8000.



DHD

DIEMOLDING HEALTHCARE DIVISION

Canastota, NY 13032 USA
(800) 847-8000 FAX (315) 697-8083
(315) 697-2221

Circle 111 on reader service card

RESPIRATORY CARE

A Monthly Science Journal. Established 1956. Official Journal of the American Association for Respiratory Care.

EDITORIAL OFFICE

11030 Ables Lane
Dallas TX 75229
(214) 243-2272

EDITOR

Pat Brougher BA RRT

ASSOCIATE EDITOR

Kaye Weber MS RRT

MANAGING EDITOR

Ray Masferrer BA RRT

EDITORIAL BOARD

James K Stoller MD, Chairman
Richard D Branson RRT
Crystal L Dunlevy EdD RRT
Charles G Durbin Jr MD
Thomas D East PhD
Dean Hess PhD RRT
Neil R MacIntyre MD
Shelley C Mishoe PhD RRT
Joseph L Ran PhD RRT

CONSULTING EDITORS

Frank E Biondo BS RRT
Howard J Birenbaum MD
Robert L Chatburn RRT
Patricia Ann Doorley MS RRT
Donald R Elton MD
Robert R Fluck Jr MS RRT
Ronald B George MD
James M Hurst MD
Charles G Irvin PhD
MS Jastremski MD
Robert M Kacmarek PhD RRT
Hugh S Mathewson MD
Michael McPeck BS RRT
David J Pierson MD
John Shigeoka MD
Jack Wanger MBA RPFT RRT
Jeffrey J Ward Med RRT

JOURNAL ASSOCIATES

Stephen M Ayres MD
Reuben M Cherniack MD
Donald F Egan MD
Gareth B Gish MS RRT
George Gregory MD
Ake Grenvik MD
H Frederick Helmholtz Jr MD
John E Hodgkin MD
William F Miller MD
Thomas L Petty MD
Alan K Pierce MD
Henning Pontoppidan MD
John W Severinghaus MD
Barry A Shapiro MD

PRODUCTION STAFF

Linda Barcus Donna Knauf
Steve Bowden Gary Caywood

CONTENTS

May 1995

Volume 40, Number 5

SPECIAL ISSUE

RESUSCITATION IN ACUTE CARE HOSPITALS

Part II

The Proceedings of a Conference

held October 21-23, 1994

in Cancun, Mexico

Chairmen and Guest Editors

Thomas A Barnes EdD RRT and Charles G Durbin Jr MD

CONFERENCE PROCEEDINGS

- 479** Ventilatory Support during Cardiopulmonary Resuscitation
by Richard D Branson and Jay A Johannigan—Cincinnati, Ohio
- 498** Methods of Emergency Drug Administration
by Dean Hess and Ravikumar Alagar—Boston, Massachusetts
- 515** Resuscitation: When is Enough, Enough?
by Robin J Hamill—Charlottesville, Virginia
- 528** Do-Not-Resuscitate Orders: A Critical Review of the Literature
by Gordon D Rubenfeld—San Francisco, California
- 538** Research on ACLS Training—Which Methods Improve Skill & Knowledge Retention?
by William Kaye—Providence, Rhode Island
- 550** Strategies for Maintaining ACLS Skills in Hospitals
by Karen M Boudin—Stanford, California
- 567** The First ACLS Course—Resuscitation Training for Students
by Robert R Fluck Jr—Syracuse, New York
- 575** Neonatal Resuscitation Program and Pediatric Advanced Life Support
by Cynthia Malinowski—Loma Linda, California

ABSTRACTS

- 462** Summaries of Pertinent Articles from Other Journals

RESPIRATORY CARE (ISSN 0020-1324) is a monthly publication of Daedalus Enterprises Inc for the American Association for Respiratory Care. Copyright © 1994 by Daedalus Enterprises Inc, 11030 Ables Lane, Dallas TX 75229. All rights reserved. Reproduction in whole or in part without the express, written permission of Daedalus Enterprises Inc is prohibited. The opinions expressed in any article or editorial are those of the author and do not necessarily reflect the views of Daedalus Enterprises Inc, the Editorial Board, or the American Association for Respiratory Care. Neither can Daedalus Enterprises Inc, the Editorial Board, or the American Association for Respiratory Care be responsible for the consequences of the clinical applications of any methods or devices described herein. Printed in USA.

RESPIRATORY CARE is indexed in Hospital Literature Index and in Cumulative Index to Nursing and Allied Health Literature.

Subscription Rates: \$5.00 per copy; \$50.00 per year (12 issues) in the US; \$70.00 in all other countries (add \$84.00 for airmail).

Second Class Postage paid at Dallas, TX. POSTMASTER: Send address changes to RESPIRATORY CARE, Daedalus Enterprises, Inc, 11030 Ables Lane, Dallas TX 75229.

PRESENTING THE FIRST PORTABLE VENTILATOR THAT DOESN'T HAVE TO APOLOGIZE FOR BEING PORTABLE!

*Respiratory Care Magazine,
January 1992, Vol. 37, No. 1.

Compare Uni-Vent™ to any other portable
ventilator and you will quickly see why
the Model 750 is in a class by itself.*



- Control, Assist-Control and SIMV operating modes, optional electronic demand valve - all PEEP compensable!
- Comprehensive alarm system and automatic continuous system self-checks for maximum safety!
- Easy-to-operate, logical control groupings, simplify personnel training!
- Operates from internal battery or external power - consumes no gas!
- High-reliability, electronic circuitry is unaffected by changes in altitude

For more information on the Uni-Vent™ Model 750, or the name of your local Representative, call Impact today!
IMPACT Instrumentation, Inc., 27 Fairfield Place, P.O. Box 508, West Caldwell, NJ 07006 1-800/969-0750
Circle 115 on reader service card

MANUSCRIPT SUBMISSION

We attempt to print Instructions for Authors and Typists near the end of *RESPIRATORY CARE* on a quarterly basis (eg, Jan, Apr, July, Nov). Call the Editorial Office for a copy if you're unable to locate.

PHOTOCOPYING & QUOTATION

PHOTOCOPYING. Any material in this journal that is copyrighted by Daedalus Enterprises Inc may be photocopied for noncommercial purposes of scientific or educational advancement.

QUOTATION. Anyone may, without permission, quote up to 500 words of material in this journal that is copyrighted by Daedalus Enterprises Inc, provided the quotation is for noncommercial use, and provided *RESPIRATORY CARE* is credited. Longer quotation requires written approval by the author and publisher.

SUBSCRIPTIONS/CHANGES OF ADDRESS

RESPIRATORY CARE
11030 Ables Lane
Dallas TX 75229-4593
(214) 243-2272

SUBSCRIPTIONS. Individual subscription rates are \$50.00 per year (12 issues) in the U.S. and Puerto Rico, \$70.00 per year in all other countries; \$95.00 for 2 years in the U.S. and Puerto Rico, \$135.00 in all other countries; and \$140 for 3 years in the U.S. and Puerto Rico, \$200.00 in all other countries (add \$84.00 per year for air mail). Annual subscriptions are offered to members of associations according to their membership enrollment as follows: 101-500 members—\$6.00, 501-1,500 members—\$5.50, 1,501-10,000 members—\$5.00, and over 10,000 members—\$4.00. Single copies, when available, cost \$5.00; add \$7.00 air mail postage to overseas countries.

CHANGE OF ADDRESS. Six weeks notice is required to effect a change of address. Note your subscription number (from the mailing label) your name, and both old and new address, including zip codes. Please note your subscription number on the envelope. Copies will not be replaced without charge unless request is received within 60 days of the mailing in the U.S. or within 90 days in other countries.

MARKETING DIRECTOR

Dale Griffiths

ADVERTISING ASSISTANT

Beth Binkley

ADVERTISING. Display advertising should be arranged with the advertising representatives. *RESPIRATORY CARE* does not publish a classified advertising column.

PRODUCT ADVERTISING:

RATES & MEDIA KITS

Gregory J Pessagno
Williams & Wilkins Adv
428 E Preston St
Baltimore MD 21202-3993
(410) 528-4218
fax (410) 528-4452

RECRUITMENT ADVERTISING:

Jason Pointe
Williams & Wilkins Adv
428 E Preston St
Baltimore MD 21202-3993
(800) 528-1843
fax (410) 528-4452

CONTENTS,

Continued

May 1995
Volume 40, Number 5

CORRECTION

- 574** Error in "Dyspnea during Weaning," published in March (Marini J. Dyspnea during weaning. (*Respir Care* 1995;40(3):271-276).

CALL FOR OPEN FORUM ABSTRACTS

- 595** 1995 Call for OPEN FORUM Abstracts

MANUSCRIPT PREPARATION GUIDE

- 588** Instructions for Authors and Typists

NOTICES

- 594** Examination Dates, Notices, Prizes

CALENDAR OF EVENTS

- 597** Meeting Dates, Locations, Themes

INDEXES

- 600** Authors in This Issue
600 Advertisers in This Issue
472 Advertiser Help Lines

Editorials and Commentaries To Note

Pulmonary and Critical Care Procedures (commentary)—J Hansen-Flaschen. *Am J Respir Crit Care Med* 1995;151:275-277. (Pertains to Tape et al paper abstracted on Page 466.)

Sleep Position and Sudden Infant Death Syndrome (editorial)—MM Willinger. *JAMA* 1995;273:818-819. (Pertains to papers by Dwyer et al and Klonoff-Cohen et al abstracted on Page 468.)

When Is CPR Futile?—A Alpers and B Lo. *JAMA* 1995;273(2):156-158. (Pertains to Curtis et al paper abstracted on Page 470.)

Prospects and Problems in Using Data from HMOs for the Study of Aging Populations and Their Health-Care Needs—ML Durham. *Gerontologist* 1995;34(4):481-485.

Inhaled Beta-Adrenergic Receptor Agonists in Asthma: More Harm than Good (commentary)—TE Barrett and BL Strom. *Am J Respir Crit Care Med* 1995;151:574-577.

Nutritional Supplementation in the ICU: How Critical Is Nutrition for the Critically Ill? (commentary)—R Koretz. *Am J Respir Crit Care Med* 1995;151:570-573.

Pulmonary Sequence of Insults to the Lungs in Early Life (review)—MEB Wohl. *Pediatr Pulmonol* 1995;19(2):90-95.

Functions of the Surfactant Proteins: A Perspective—S Hagwood and FR Poulain. *Pediatr Pulmonol* 1995;19(2):99-104.

Cardiorespiratory Adaptation during Sleep in Infants and Children—C Gaultier. *Pediatr Pulmonol* 1995;19(2):105-117.

Assessment of Lung Function in the Intensive Care Unit—AH Nama and CJL Newth. *Pediatr Pulmonol* 1995;19(2):118-128.

Lack of Association between Atelectasis and Fever—M Engoren. *Chest* 1995;107:81.

Postoperative fever occurs in many patients. If no infection is found, atelectasis, if present, may be blamed. This study of 100 postoperative cardiac surgery patients followed up from day of surgery through the second postoperative day with daily portable chest radiographs and continuous bladder thermometry was designed to look for an association between atelectasis and fever. The daily incidence of atelectasis increased from 43 to 69 to 79%. However, the incidence of fever, defined as temperature $\geq 38.0^\circ\text{C}$ fell from 37 to 21 to 17%. When defined as temperature $\geq 38.5^\circ\text{C}$, the daily incidence of fever fell daily from 14 to 3 to 1%. Using χ^2 analysis, no association could be found between fever and amount of atelectasis. This contradicts common textbook dogma but agrees with previous human study and animal experiments.

Intraindividual Peak Flow Variability—MJ Hegewald, RO Crapo, RL Jensen. *Chest* 1995;107:156.

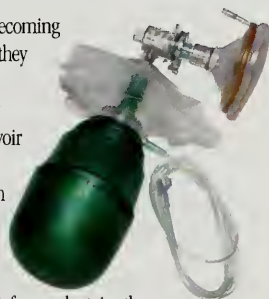
OBJECTIVES: To quantify intraindividual variability in peak expiratory flow (PEF) measured with peak flowmeters and to define factors affecting PEF variability. **METHODS:** Three hundred one healthy subjects (aged 4 to 84 years) were recruited from sites at sea level ($n = 220$) and at 1,400 m altitude ($n = 81$). All testing was done with the same model peak flowmeter. Each subject was actively coached through five to eight successive PEF maneuvers. Three meters of the same model were tested using a mechanical waveform simulator at three different flows at both testing altitudes (sea level and at 1,400 m). **RESULTS:** Excluding outliers, the mean PEF was 523 L/min, mean standard variation (SD) was 22 L/min, and mean coefficient of variation (CV) was 4.6%. The upper 95th percentile for CV

was 8% for adults and 10% for youths. Analyzing only the three highest peak flows for each subject, the mean PEF was 539 L/min, mean SD was 12 L/min, and mean CV was 2.4%. The upper 95th percentile for CV was 6% for adults and 9% for youths. Linear regression analysis revealed a small but statistically significant correlation ($p < 0.01$) between mean peak flow and CV. In adults, SD correlated with sex ($p < 0.01$) but neither CV nor SD was correlated with age, height, weight, or altitude. Meter variability defined with the mechanical waveform simulator was small. Standard deviation varied from 1.5 to 4.2 L/min and CV from 0.4 to 1.6%. When the three largest peak flows for each subject were used, 5.5% of intraindividual variance was explained by meter variance. **CONCLUSIONS:** These estimates of intraindividual variability in healthy subjects are generally lower than those previously reported. Meter variability accounts for only a small part of total intraindividual variability. The 95th



There comes a time when all the valuable features become invaluable

BagEasy® Disposable Manual Resuscitators have a way of becoming essential to the people who use them. Some say it's because they can use PEEP without first searching for a separate valve. BagEasy's PEEP valve is built in. Others say it's because they know exactly when to increase flow. BagEasy's oxygen reservoir is sensibly placed up front, where you can see it. Still others find BagEasy remarkable because it lets them change position in a crowded space without interrupting flow. BagEasy has two 360° swivels and a flexible front end. We expect some will find BagEasy essential because of our Conformable Circle Seal® mask. It conforms to the contour of the patient's face and retains the shape for an easy seal. BagEasy comes in adult, child, and infant sizes. Find out what makes it invaluable to you.



RESPIRONICS INC.

THE FIRST NAME IN INNOVATIVE RESPIRATORY CARE



1001 Murry Ridge Drive • Murrysville, Pennsylvania 15668-8550 USA
Phone: 1-800-345-6443 within the USA and Canada or 1-412-733-0200 • Fax: 1-412-733-0299

percentile data suggest that a fall in PEF of 6 to 8% in adults and 9 to 10% in youths would be statistically significant.

Work of Breathing after Extubation—AM Issaaya, SD Nathan, MJ Belman. *Chest* 1995;107:204.

Recently we showed that work of breathing was higher in the immediate period after extubation as compared with spontaneous breathing through an endotracheal tube. In this study, we evaluated the glottis and trachea as potential sites of increased airway resistance after extubation. We measured breathing pattern, work of breathing, and pressure time product in eight patients during weaning from mechanical ventilation. We acquired data during pressure support ventilation and spontaneous breathing via the ventilator, with the endotracheal tube in place, and after extubation. During bronchoscopy at the time of extubation, we examined the trachea and measured the cross-sectional area of the glottis. Work of breathing and pressure time product were significantly lower during pressure support ventilation as compared with spontaneous breathing after extubation (0.43 ± 0.10 vs 1.49 ± 0.10 J/L and 101 ± 22 vs 299 ± 30 cm H₂O · s/min, respectively; $p < 0.05$). However, both indexes were significantly higher after extubation as compared with breathing through the endotracheal tube (1.49 ± 0.10 vs 0.95 ± 0.12 J/L, 299 ± 31 vs 196 ± 26 cm H₂O · s/min respectively; $p < 0.05$). During bronchoscopy, no tracheal or glottic narrowing was detected. The glottic cross-sectional area was successfully measured in 4 patients at the onset of inspiration and found to be 140 ± 15 mm². This value was larger than the mean cross-sectional area of the endotracheal tubes used in these patients (50 mm²). We conclude that neither tracheal nor laryngeal disease caused the increase in work of breathing after extubation. Our data suggest that upper airway narrowing at a more proximal site, such as the oropharynx or velopharynx may be the cause of the increase in respiratory work.

Knowledge about Pulse Oximetry among Medical and Nursing Staff—MD Stoneham, GM Saville, JH Wilson. *Lancet* 1994; 344:1339.

Although pulse oximeters are increasingly used to monitor patients on medical and surgical wards, staff seem to receive no specific training in the operation of these devices. We investigated the knowledge of junior doctors and nursing staff about pulse oximetry as used on seriously ill patients in a District-General Hospital in Exeter UK. Thirty medical or surgical pre-registration house officers or senior house officers and 30 staff nurses answered a structured

questionnaire. Questions were asked about the theory behind pulse oximetry, factors affecting readings, "normal" values in various patients, values in hypothetical clinical situations, and what training subjects had received. Responses were scored against standard answers from reference texts. 97% of doctors and nurses did not understand how a pulse oximeter worked and were confused about factors influencing readings. Respondents gave a wide range of acceptable saturation values (eg. 90-100% for a fit adult), thus demonstrating poor understanding of physiological principles. There were serious errors made in evaluating saturation readings in hypothetical clinical situations. Only 1 doctor had received formal training in the use of pulse oximetry. Our survey revealed that junior doctors and staff nurses were untrained in pulse oximetry, lacked knowledge of basic principles, and made serious errors in interpretation of readings. Training is needed for staff who use pulse oximeters.

Clinical Risks for Development of the Acute Respiratory Distress Syndrome—LD Hudson, JA Milberg, D Anardi, and RJ Maunder. *Am J Respir Crit Care Med* 1995;151:293.

To further understanding of the epidemiology of acute respiratory distress syndrome (ARDS), we prospectively identified 695 patients admitted to our intensive care units from 1983 through 1985 meeting criteria for seven clinical risks, and followed them for development of ARDS and eventual outcome. ARDS occurred in 179 of the 695 patients (26%). The highest incidence of ARDS occurred in patients with sepsis syndrome (75 of 176, 43%) and those with multiple emergency transfusions (≥ 15 units in 24 h) (46 of 115, 40%). Of patients with multiple trauma, 69 of 271 (25%) developed ARDS. If any two clinical risks for trauma were present, the incidence of ARDS was 23 of 57, or 40%. During the study period, we identified 48 patients with ARDS who did not have one of the defined clinical risks, yielding a sensitivity of 79% (179 of 227). Secondary factors associated with increased risk for ARDS in clinical risk subgroups include an elevated Acute Physiologic and Chronic Health Evaluation II (APACHE II) score in patients with sepsis and increased APACHE II and Injury Severity Scores (ISS) in trauma victims. Mortality was threefold higher when ARDS was present (62%) than among patients with clinical risks who did not develop ARDS (19%; $p < 0.05$). The difference in mortality if ARDS developed was particularly striking in patients with trauma (56% versus 13%), but less in those with sepsis (69% versus 49%). The mortality data should be interpreted with caution, since the fatality rate in ARDS patients appears to have decreased in our institution from the time that these data were collected.

These findings should be considered in the design of studies aimed at preventing or modifying the development of ARDS.

Assessment of the Severity of Asthma by an Expert System: Description and Evaluation—H Redier, J-P Daurès, C Michel, H Proudhon, D Vervloet, D Charpin, J Marsac, D Dussier, C Brambilla, B Wallaert, M-C Kopperschmitt, G Pauli, A Tayart, O Cogis, F-B Michel and P Godard. *Am J Respir Crit Care Med* 1995;151:345.

Asthmaexpert, an expert system (ES), was produced at the special request of several clinicians in order to better understand the medical decisions made by clinical experts in managing an asthmatic patient. We describe and evaluate this knowledge base, focusing mainly on assessment of the severity of asthma. After compiling data from a patient, Asthmaexpert assesses the severity of the disease and identifies the trigger factors involved, suggests any further investigations that may be required, and offers a treatment strategy. Implemented with Nexpert and Hypercard, it runs on a Macintosh personal computer. The validation stage involved eight clinical experts who provided 20 case report forms (CRF) with their conclusions about management of asthma. The CRF were then programmed into the ES, which provided its own conclusions about the same subjects. Afterward, all the experts evaluated the conclusions given by ES or by their colleagues in a double-blind manner. One hundred twenty-seven CRF were available. The reliability of the experts' opinions was good, with a substantial consensus between them when assessing severity scores ($\kappa = 0.27$ to 0.54). There was no difference in concordance of opinions on severity scores either between the experts who designed the system and ES or between the other experts and ES (weighted $\kappa = 0.72$ and 0.69 , respectively). Experts judged that the severity scores given by ES were as good as those proposed by their colleagues, and that the overall conclusions given by ES were as good as or better than those given by their colleagues. The conclusions drawn by ES were given a good rating. Consequently, Asthmaexpert, which has been a useful tool for discussing management of asthma, may also be useful for teaching purposes.

Heliox Improves Pulsus Paradoxus and Peak Expiratory Flow in Nonintubated Patients with Severe Asthma—CA Manthous, JB Hall, A Melmed, MA Caputo, J Walter, JM Klocksieben, GA Schmidt, and LDH Wood. *Am J Respir Crit Care Med* 1995;151:310.

Heliox is a blend of helium and oxygen with a gas density less than that of air that decreases

New Press-and- Release Action

Simplified design, with fewer moving parts, makes canister actuation easier, minimizes need for coordination.



In MDI Drug Delivery... The choice just got easier.

Better delivery.

OptiHaler® is designed to give your patients the full benefit of their MDI medication. Its patented aerodynamic action* creates a measurably superior aerosol mixture — richer in the smaller, more effective particles.¹ And it makes more of that mixture available for delivery to the lungs than conventional holding chambers.¹

Better design.

OptiHaler's new simplified design makes MDI therapy easier than ever. Patients simply press and release the canister while inhaling — then close the end cap for the next puff. Cleaning and maintenance are easier too.

Better compliance.

And because more convenience usually means better compliance, OptiHaler packs all of these advantages into a sleek, discreet, truly portable unit that patients rate significantly more convenient for daily use than the leading holding chambers.²



New, more convenient,
one-piece cap.

So why not make MDI therapy easier for your patients...with the product more patients prefer.² Call our 24-hour customer service hotline today for your free professional sample of new, improved OptiHaler.

1-800-962-1266

NEW IMPROVED



OptiHaler®

Drug Delivery System for use with Metered Dose Inhalers

The promise of MDI therapy...delivered.

From the makers of ASSESS® and Personal Best® Peak Flow Meters
HealthScan Products Inc., Cedar Grove, NJ

REFERENCES: 1. Data on file: HealthScan Products Inc. 2. Wheeler BB, Bollers ND: Initial experience with a novel spacer device. (Presented at the 1993 meeting of the American Academy of Allergy and Immunology). * U.S. Patent No. 5,040,527 OA760004-0 © 1994, HealthScan Products Inc.

airway resistance (R_{aw}) in patients ventilated for status asthmaticus. We tested whether breathing an 80:20 mixture of helium:oxygen would reduce pulsus paradoxus (PP) and increase peak expiratory flow (PEF) in patients presenting to the emergency room with an exacerbation of asthma. After receiving 30 min of β -agonist aerosols and intravenously administered methylprednisolone, 27 patients whose PP remained greater than 15 mm Hg and whose PEF remained less than 250 L/min consented to breathe heliox or room air for 15 min. PP decreased and PEF increased with time in control patients, indicating a time related effect of routine bronchodilator therapy ($p < 0.05$). PP decreased in 15 of 16 patients during heliox, and the change with heliox was significantly greater than that during air breathing ($p < 0.01$). PEF measured with a Wright's peak flow meter calibrated for heliox increased in all patients breathing heliox. Again, the increase in PEF during heliox breathing was significantly greater than the corresponding change in control patients breathing air ($p < 0.001$). To the extent that PP reflects the inspiratory fall in pleural pressure, this reduction in PP indicates a substantial reduction in inspiratory R_{aw} when the less dense gas is inspired through narrowed bronchi having turbulent flow regimes. The 35% increase in PEF while breathing heliox signals a similar reduction in expiratory R_{aw} , which might diminish the hyperinflation often observed during an exacerbation of asthma. Taken together, these effects of heliox are likely to diminish the tendency to inspiratory muscle fatigue until bronchodilation is effected during initial treatment of severe asthma.

Asthma Self-Management Education Program by Home Monitoring of Peak Expiratory Flow—JM Ignacio-Garcia and P Gonzalez-Santos. *Am J Respir Crit Care Med* 1995;151:353.

A prospective controlled trial of home monitoring of peak expiratory flow rate (PEFR) was conducted to determine the usefulness of an objective measure of lung function in association with an education program and a medication self-management plan in reducing morbidity in adult patients with asthma. Thirty-five patients managed themselves, using peak flow readings as the basis for the therapeutic plan coupled with educational intervention, whereas 35 control patients used symptoms and spirometric data for following physicians' treatment plans. After a 6-month study period, patients in the experimental group showed statistically significant improvements in morbidity parameters (days lost from work, acute asthma attacks, days on antibiotic therapy, physician consultations,

and emergency room admissions for asthma), increases in FVC, FEV₁, and FEV₁/FVC, mean PEF and mean morning PEF, decrease in percentage of the mean PEF amplitude, and a reduction in the use of inhaled β -agonists, oral theophylline, and oral prednisone. Although improvements in some of these parameters were also found in the control group, they did not reach the levels of significance obtained in the experimental group. The personal use of an objective measure of lung function in association with a medication self-management plan leads to improvement in the patient's condition.

Efficacy of Uniphy®[®], Salbutamol, and Their Combination in Asthmatic Patients on High-Dose Inhaled Steroids—RN Rivington, L-P Boullet, J Côté, H Kreisman, DI Small, M Alexander, A Day, Z Harsanyi, and AC Darke. *Am J Respir Crit Care Med* 1995;151:325.

A group of 32 patients with moderately severe, chronic asthma (mean FEV₁ 55% of predicted), maintained on moderately high doses of inhaled corticosteroids (mean dose 1,100 μ g/dL), participated in this doubleblind, placebo-controlled crossover study. The effect on pulmonary function of adding theophylline (U, once daily Uniphy®[®]), inhaled salbutamol (S, 200 μ g four times per day), and their combination (C) or placebo (P) was assessed on Day 14 of each treatment phase. Patients recorded peak expiratory flow, asthma symptom severity (morning and evening), and use of rescue salbutamol inhaler in daily diaries. Mean FEV₁ between 0730 and 1800 h and maximum FEV₁ between 0730 and 1300 h were significantly higher on U, S, and C compared with P ($p < 0.006$). Morning peak flow and FEV₁ (0730 h) were significantly higher on U and C compared with S and P ($p < 0.01$). Evening peak flow was higher on U than P ($p < 0.001$), and C was higher than S and P ($p < 0.01$). Rescue salbutamol inhaler use was significantly higher on P than on U, C, or S ($p = 0.0001$). Patient rating of asthma symptoms during C was significantly better than on S or P ($p < 0.05$). Patient rating of asthma control and study phase preference was significantly higher on combination and Uniphy alone than on placebo, the combination also being superior to salbutamol alone. Addition of Uniphy or a combination of Uniphy and salbutamol significantly improves pulmonary function and asthma symptoms in patients treated with high doses of inhaled corticosteroids and as-needed β -agonists. These findings are supportive of the international guidelines for the treatment of asthma.

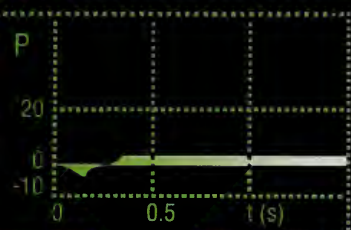
Procedural Skills of Practicing Pulmonologists: A National Survey of 1,000 Members of the American College of Physicians—TG Tape, LL Blank and RS Wigton. *Am J Respir Crit Care Med* 1995;151:282.

We surveyed pulmonologists to determine which procedures they do in practice, where they learned the procedures, and how much training they recommend to attain and maintain clinical competence in each. We mailed a survey to a random sample of 1,000 members of the American College of Physicians who were identified as practicing pulmonologists; 755 (75%) responded. Respondents performed a variety of pulmonary procedures, an average of 17 of the 29 listed. Pulmonologists who were more recent graduates, who worked longer hours, and who were involved in critical care did a greater variety of procedures. Only 26% of practicing pulmonologists currently do all the procedures required for board certification in pulmonary medicine. For each of 13 specific procedures, the number reported done in the past year was generally unrelated to practice factors. Many respondents who learned procedures in practice did so without formal training or supervision. Respondents' recommendations regarding numbers of procedures required to attain or maintain competence did not vary greatly. Pulmonologists vary considerably in the types of procedures they do. Their opinions about the training needed for competence help to better define requirements for training programs. More attention should be focused on training and certifying practicing pulmonologists in procedures learned after formal fellowship training.

Gender Differences in Lung Growth—M Hibbert, A Lannigan, J Raven, L Landau, and P Phelan. *Pediatr Pulmonol* 1995;19:129.

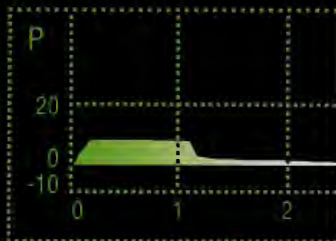
Annual measurements of lung volumes and forced expiratory flows were made in 281 boys and girls from 8 to 12 years and in another cohort of 287 from 12 to 20 years to measure longitudinal lung growth. Gender differences in growth of lung function were documented, with girls generating greater volume-standardized maximal expiratory flows until age 18.5 years. Beyond that age, boys generated higher expiratory flows in proportion to total lung capacity (TLC). There was a time lag of up to 1 year between the age of peak growth velocity in lung volume and peak growth velocity in height. Age at peak growth in flow lagged another year behind that in volume. This was noted more in boys than girls. Dysanaptic lung growth was found with differing rates of growth of maximal expiratory flow compared with TLC or vital capacity (VC).

CPAP



Occlusion-pressure

CMV



Intrinsic PEEP

Always A Step Ahead!

Evita makes work of breathing even easier!

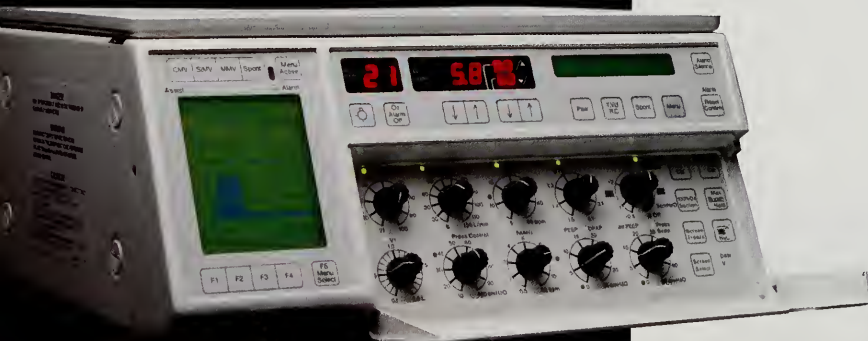
Now Dräger provides you with two more powerful tools to optimize weaning of your patients.

Introducing **Flowtrigger** without increase of expiratory resistance, combined with **P01 measurement** to determine the patients ventilatory drive.

To extend monitoring capabilities Evita now includes the ability to measure **Intrinsic PEEP** with the display of **Trapped Volume**.

With Dräger you can stay one step ahead in providing safe, patient friendly ventilation. For the difference your patients can feel, choose...

Dräger: Technology for Life



Flowtrigger

Dräger
Technology for life

4101 Pleasant Valley Road Suite 100 Chantilly, VA 22021
Tel (703) 817-0100 Fax (703) 817-0101

Circle 101 on reader service card

Clinical Value of Antenatal Fetal Pulse Oximetry—E van Oudgaarden and N Johnson. *J Perinat Med* 1994;22:295.

The aim of this prospective observational study was to see if high quality fetal pulse oximetry signals can be obtained from the fetus before labour. It was carried out in Labour Ward, Clarendon Wing, Leeds General Infirmary and St James Hospital, Leeds. We studied Caucasian term fetuses antenatally and 20 fetuses during labour. Antenatal fetal oxygen saturation was measured using a sensor passed through the cervix onto the membranes covering the fetus before the onset of labour. The quality of these signals was compared to data obtained from 20 fetuses monitored during active labour after amniotomy. High quality pulse oximetry signals were obtained for less than 1% of the monitoring time before the onset of labour (95% CI, 0-2.5%). High quality data were seen 38.6% of the time once the membranes were ruptured and the head descended into the pelvis. With present technology, fetal pulse oximetry will not contribute to antenatal fetal assignment.

Blue-collar Normative Spirometric Values for Caucasian and African-American Men and Women Aged 18 to 65—HW Glindmeyer, JJ Lafante, C McCollister, RN Jones, and H Weill. *Am J Respir Crit Care Med* 1995;151:412.

Normative spirometric values were derived from 5,042 white (of mainly European ancestry) and black (of mainly African ancestry) men and women paper plant workers who are never-smokers, with no respiratory symptoms or diagnoses and no history of occupational exposure to fibrogenic dusts or irritant chemicals. This cohort was selected from a much larger population under long-term respiratory surveillance ($n > 50,000$ at 50 plants). Standardized equipment, procedures, and data reduction methods complied with ATS recommendations. Data were collected by the medical departments of the participating companies as part of their routine health surveillance, and the graphic and numeric test results were transmitted to the Tulane University Section of Environmental Medicine for centralized quality assurance, interpretation, and archiving. The large numbers allow derivation of gender- and race-specific reference values. Lower limits of normal were derived and depend upon residual variation and any changes in variation with age. The results indicate that polynomial regression equations provide a significantly better fit than linear regressions with breakpoints. In addition to being more biologically plausible, the polynomial model more closely matches observed longitudinal changes in lung function with age. The age range of the cohort, 18 to 65, provides a regression that more closely

matches the observed values in this range, because it does not include "supernormal" elderly survivors, which can lessen the slope of the regression and artifactually increase the predicted values of 50 to 65 yr olds. The regression equations derived for black men and women do not support the use of a single race adjustment (0.85 or 0.88) for all age, sex, height, and spirometric test parameter combinations. These race- and gender-specific regression equations, with their respective lower limits of normal, should improve the detection and quantification of adverse health effects in working individuals and populations.

The Contribution of Changes in the Prevalence of Prone Sleeping Position to the Decline in Sudden Infant Death Syndrome in Tasmania—T Dwyer, A-L Ponsosby, L Blizard, NM Newman, JA Cochrane. *JAMA* 1995;273:783.

Objective: To determine the independent contribution of changes in infant sleep position to the recent decline in sudden infant death syndrome (SIDS) rate in Tasmania. **Design:** (1) A comparison of the whole population incidence of SIDS before and after an intervention to reduce the prevalence of prone sleeping position. (2) A within-cohort analysis of the contribution of sleep position and other exposures to the decline in SIDS after the intervention. **Setting:** Tasmania, Australia. **Participants:** (1) All SIDS cases from 1975 through 1992. (2) A sample of one in five infants born in Tasmania who at perinatal assessment were scored to be at higher risk for SIDS since January 1988. Of 5,534 infants included in the study, 39 later died of SIDS. **Interventions:** Multiple public health activities to reduce the prevalence of the prone infant sleeping position in Tasmania and verbal information on the association between prone position and SIDS to cohort participants from May 1, 1991. **Main Outcome Measure:** Sudden infant death syndrome incidence. **Results:** The Tasmanian SIDS rate decreased ($P < .01$) from 3.8 (95% confidence interval [CI], 3.5 - 4.2) deaths per 1,000 live births from 1975 through 1990 to a rate of 1.5 (95% CI, 0.9 - 2.2) deaths per 1,000 live births in 1991 through 1992. The SIDS mortality rate in the cohort by period of birth was 7.6 (95% CI, 4.9 - 10.3) deaths per 1,000 live births for those born from May 1, 1988, through April 30, 1991, and 4.1 (95% CI, 1.3 - 7.0) deaths per 1,000 infants for those born from May 1, 1991, through October 31, 1992. The prevalence of usual prone sleeping position at 1 month of age was 29.9% and 4.3% in these two cohorts, respectively (adjusted odds ratio, 0.11; 95% CI, 0.08 - 0.13). Logistic regression demonstrated that 70% of the SIDS rate reduction in the cohort could be accounted for by the decreased prevalence of the prone sleeping position. Other factors examined indi-

vidually contributed to less than 10% of the SIDS rate reduction. **Conclusions:** The major contributing factor to the recent SIDS rate decline in Tasmania has been the reduction in the proportion of infants usually sleeping prone.

A Case-Control Study of Routine and Death Scene Sleep Position and Sudden Infant Death Syndrome in Southern California—HS Klonoff-Cohen. *JAMA* 1995;273:790.

Objective: To investigate whether infants who died of sudden infant death syndrome (SIDS) were routinely placed in different sleep positions compared with healthy infants in a multi-ethnic diverse population in the United States. **Design:** A population-based case-control study. **Setting:** Five counties in Southern California including Los Angeles, Orange, San Bernardino, Riverside, and San Diego. **Participants:** Parents of 200 white, African-American, Hispanic, and Asian infants who died of SIDS between January 1989 and December 1992 and parents of 200 healthy, living infants matched on the basis of birth hospital, birth date, race, and gender. **Information was obtained from detailed telephone interviews with the parents and validated with obstetric and pediatric records. Main Outcome Measures:** Routine sleep position, type of bedding, and objects in bed were determined for both case and control infants, while the last-placed and found sleep and face positions at death were reported for SIDS infants. **Results:** Approximately 66% of SIDS infants and 64% of comparison infants routinely slept on their abdomens ($P = .91$). At the time of death, 80% of cases were found sleeping on their abdomens. There was no difference in routine sleep position for SIDS infants and comparison infants (odds ratio = 0.76; 95% confidence interval, 0.42 to 1.38), while simultaneously adjusting for birth weight (in grams), medical conditions at birth, breast-feeding, passive smoking, maternal recreational drug use, prenatal care, and infant vomiting. Hispanic parents routinely placed their infants on their abdomens less frequently than white parents ($P < .01$). However, the prone sleep position (face down) was the most commonly found sleep position at death in both Hispanic and non Hispanic infants. **Conclusions:** Routine prone sleep position was not associated with an increased risk of SIDS in this study population. The results need to be confirmed with other parents of SIDS infants interviewed before the height of publicity regarding prone sleep position in the United States.

The Effect of Passive Smoking and Tobacco Exposure through Breast Milk on Sudden Infant Death Syndrome—HS Klonoff-Cohen, SL Edelstein, ES Lefkowitz, IP Srinivasan, D Kaegi, JC Chang, and K Wiley. *JAMA* 1995;273:795.



Make your arrhythmia training as simple as watching TV.

- Arrhythmia Recognition
- ACLS Courses
- Defibrillation Training
- EMT-D Training and Certification Programs
- Hemodynamic Waveform Recognition
- PALS Courses

Build your own economical training system with modular components from Armstrong.

The **Armstrong Video Adapter** displays ECG and BP waveforms on television. Now ACLS, arrhythmia recognition and defibrillation training programs can be easily presented to a large group. Unique features include:

ZOOM, CASCADE, SCALE, FREEZE, AND CURSOR. Compatible with Armstrong Patient Simulators.

Patient Simulators help master complete ECG/arrhythmia recognition, interpretation and defibrillation. Three models to choose from, ranging from simple arrhythmia simulation to complex pressures. Many optional personality modules available, including 12-Lead ECG and Pediatric.

Chris Clean® CPR Manikin with Defib Chest is built to withstand high energy discharges from the defibrillator up to 400 joules. Compatible with

all Automatic, Semi-Automatic and Manual External Defibrillators and Darox/R2 System. Optional, reusable Defib Pad Adapters offer the flexibility of training on more than one type of defibrillator. Chris Clean has individual mouth/nose pieces and air head bags for protection against cross-contamination.



NEW Monitor/VCR Video Viewer



**Armstrong
Medical**
INDUSTRIES INC.

P.O. Box 700 • Lincolnshire, IL 60069-0700 ©1995

Call for a Free Catalog
800/323-4220
Western States: 800/442-6991

Objective: To examine the relationship between sudden infant death syndrome (SIDS) and smoking during pregnancy; postnatal tobacco smoke exposure from the mother, father, live-in adults, and day care providers; and postnatal smoke exposure from breast-feeding. **Design:** Case-control study. **Setting:** Five counties in Southern California. **Participants:** A total of 200 white, African-American, Hispanic, and Asian parents of infants who died of SIDS between 1989 and 1992 were compared with 200 control parents who delivered healthy infants. Case infants were matched to control infants on the basis of birth hospital, birth date, gender, and race. All information was obtained from a detailed telephone interview and validated with medical records. **Main Outcome Measures:** Risk of SIDS associated with passive smoking by the mother, father, live-in adults, and day care providers; smoking in the same room as the infant; total number of cigarettes smoked by all adults; and maternal smoking during the time period of breast-feeding. **Results:** Conditional logistic regression resulted in overall adjusted odds ratios (ORs) for SIDS associated with passive smoke from the mother of 2.28, the father of 3.46, other live-in adults of 2.18, and all sources of 3.50 (95% confidence interval, 1.81-6.75), while simultaneously adjusting for birth weight, sleep position, prenatal care, medical conditions at birth, breast-feeding, and maternal smoking during pregnancy. A dose-response effect was noted for SIDS associated with increasing numbers of cigarettes, as well as total number of smokers. Breast-feeding was protective for SIDS among nonsmokers (OR = 0.37) but not smokers (OR = 1.38), when adjusting for potential confounders. **Conclusions:** Passive smoking in the same room as the infant increases the risk for SIDS. Physicians should educate new and prospective parents about the risks of tobacco smoke exposure during pregnancy and the first year of the infant's life.

Continuous Versus Bilevel Positive Airway Pressure for Obstructive Sleep Apnea—MK Reeves-Hoché, DW Hudgel, R Meek, R Witteman, A Ross, and CW Zwillich. *Am J Respir Crit Care Med* 1995;151:443.

Recent objective studies demonstrate relatively low hours of nightly use during nasal continuous positive airway pressure (CPAP) therapy for obstructive sleep apnea (OSA). Patients frequently complain of dyspnea or discomfort during CPAP use, especially during expiration (against the continuous pressure), which may be a reason for the low hours of use. We hypothesized that with decreased expiratory pressure, hours of nightly use would increase. Therefore, we randomized 83 OSA patients to receive either continuous or bilevel positive

airway pressure when expiratory pressure is lower. To document objectively the effective use of either therapy, we built and installed elapsed-time and mask pressure sensors in the patients' positive airway pressure units. A total of 62 patients were evaluable and followed for 1 year. Of these, 26 received bilevel and 36 CPAP pressures. The machine timers measured accumulated "machine-on" time, and the mask pressure sensor recorded the total time in which the mask pressure was within 2 cm H₂O of the effective pressure (pressure shown to eliminate 95% of the obstructive apneas during a full night of polysomnography). The mean machine timer hours of CPAP were 5.0 ± 0.19 SEM and 4.9 ± 0.23 SEM during bilevel therapy ($p = \text{NS}$) over a 12-mo period. The pressures required during CPAP or bilevel therapy were not different between high and low hourly users. Effective use, the percentage of time that the machine was running and the prescribed pressure was being delivered was 80% in CPAP and 82% in the bilevel users ($p = \text{NS}$). Both groups had equal complaints with regard to mask discomfort, machine noise, and nasal stuffiness. In conclusion, no difference in average hours of use during 12 mo of CPAP and bilevel therapy was identified in the OSA patients studied.

Psychosocial Functioning of Young Adults with Cystic Fibrosis and Their Families—C Blair, A Cull, and CP Freeman. *Thorax* 1994;49 (8):798.

Background: The psychosocial functioning of adolescents and young adults with cystic fibrosis still living in the parental home was investigated. With its proven genetic aetiology cystic fibrosis is an ideal model with which to assess the impact of a chronic and life threatening disorder on family and individual psychological and social functioning. **Methods:** Twenty nine patients with cystic fibrosis and their families were compared with those of 27 patients with anorexia nervosa and 31 well controls. Assessments were made using self reporting, interview, and observational methods. **Results:** Most patients with cystic fibrosis were in robust psychological health and only differed from their healthy peers in that they were much less likely to be in employment. Mothers of patients with cystic fibrosis or anorexia nervosa were more likely than the mothers of the well group to be emotionally distressed, although this was not so for fathers. Young people in both illness groups were more likely to have parents with high levels of expressed emotion. Most families of patients with cystic fibrosis had good problem solving abilities. **Conclusions:** In spite of the burden of illness in cystic fibrosis psychological functioning in many respects matches that of well young people.

Use of the Medical Futility Rationale in Do-Not-Attempt-Resuscitation Orders—JR Curtis, DR Park, MR Krone, RA Pearlman. *JAMA* 1995; 273: 124.

Objective: To describe the use of the medical futility rationale in do-not-attempt-resuscitation (DNAR) orders written for medical inpatients. **Design:** Structured interviews with medical residents. **Methods:** Second- and third-year internal medicine residents ($n = 44$) were telephoned weekly and briefly interviewed about each patient who received a DNAR order in the preceding week. **Setting:** Two university-affiliated hospitals: a veterans affairs medical center and a municipal hospital. **Patients:** One hundred forty-five medical inpatients for whom DNAR orders were written during their hospitalization. **Results:** Residents stated that the medical futility rationale applied for 91 patients (63%), but this rationale was invoked independent of patient or surrogate choice for only 17 patients (12%). Of the 91 patients for whom futility applied, both quantitative futility (low probability of success) and qualitative futility (poor quality of life if cardiopulmonary resuscitation [CPR] were performed) applied to 45 (49%), quantitative futility alone to 30 (33%), and qualitative futility alone to 16 (18%). Residents report that they discussed resuscitation issues with all communicative patients for whom the medical futility rationale was invoked. Among patients for whom quantitative futility applied, residents' predictions of the probability that patients would survive to hospital discharge after CPR varied from 0% (for 60% of patients) to 75%. For 32% of these patients, residents predicted survival at 5% or more. Logistic regression modeling showed that the presence of organ failure (odds ratio [OR], 8.9; 95% confidence interval [CI], 3.3-23.9), the residents' estimates of the probability of surviving CPR (OR, 0.94; 95% CI, 0.88-0.99), and nonwhite race (OR, 2.7; 95% CI, 1.1-6.3) were associated with the determination of quantitative futility. In one third of the cases where qualitative futility applied, residents made the judgment of qualitative futility without discussing quality of life with communicative patients. Logistic regression modeling showed immobility (OR, 3.2; 95% CI, 1.1-9.0) and age ≥ 75 years (OR, 0.3; 95% CI, 0.1-0.8) to be associated with the determination of qualitative futility. **Conclusions:** While residents did not appear to use the medical futility rationale to avoid discussing DNAR issues with patients, we found evidence of important misunderstandings of the concepts of both quantitative and qualitative futility. If the futility rationale is to be applied to withholding or withdrawing medical interventions, practice guidelines for its use should be developed, and education about medical futility must be incorporated

Multiple Choice

REMstar Choice® Nasal CPAP System

REMstar Choice is the therapy of choice for OSA disorders. It's the first – and only – CPAP therapy that combines a patient specific pressure ramp function with a wireless remote control for patient use. And it is the only unit to offer so many excellent choices...



CHOICE: To operate REMstar Choice, patients can use an exclusive wireless remote or the controls on the unit.

CHOICE: Patients can activate the pressure ramp at any time by touching a button on the remote control or on the unit itself.



CHOICE: For patient safety, physicians determine the maximum ramp time – between 0 - 45 minutes – based upon the patient's needs.

CHOICE: Optional easy-to-use pass-over humidifier relieves nasal dryness.

CHOICE: Optional tubing and remote control convenience gives patients the choice of placing the unit up to 12 feet from the bed.

***The world's most popular CPAP system
now comes with a two-year warranty
(third year available).***



CHOICE: Now you can offer the best Choice in CPAP technology. For details, call our customer service department at (800) 345-6443.



RESPIRONICS INC.®

THE FIRST NAME IN INNOVATIVE RESPIRATORY CARE

1001 Murry Ridge Drive, Murrysville, Pennsylvania 15668-8550 USA Phone: (800) 345-6443 within the USA and Canada or (412) 733-0200 Fax: (412) 733-0299

©Respironics Inc. 1993

Circle 129 on reader service card

into medical school, residency training, and continuing medical education programs.

Spirometric Reference Values for White European Children and Adolescents: Polgar Revisited—PH Qunjer, GJM Borsboom, B Brunkreef, M Zach, G Forche, JE Cotes, J Sanchis, and P Paoletti. *Pediatr Pulmonol* 1995;19:135.

Summary: We analyzed six spirometric data sets collected in the Netherlands, Austria, the United Kingdom, Spain, and Italy. The objectives were to establish whether (1) it was possible to describe spirometric indices from childhood to adulthood, taking into account the adolescent growth spurt, and (2) there are systematic differences in ventilatory function between children and adolescents in different parts of Western Europe. The study comprised 2,269 girls and 3,592 boys, aged 6-21 years. The range in standing height was 110-185 cm, in girls, 110-205 cm, in boys. The model applicable to all data sets was $\ln FVC$ or $\ln FEV_1 = a + (b + c \cdot A) \cdot H$, where H = standing height and A = age; this model prevents the phase shift between the adolescent growth spurt in length and lung volume from leading to an age-dependent bias in predicted values. There was surprising agreement between most of the data sets; systematic differences are probably due to technical factors arising from ATPS-BTPS corrections and from defining the end of breath with pneumotachometer systems. Taking those into account, prediction equations for FVC , FEV_1 , and $FEV_1\%FVC$ were developed with "lower limits of normal" which should be applicable to children and adolescents of European descent. It is proposed that the approach of analyzing available data sets should also be applied to other ventilatory indices, data collected in adults and elderly subjects, or in other ethnic groups, and that an international database be set up to that end.

**Don't miss the
Research Symposium
at the 1995
ANNUAL CONVENTION
of the American
Association for
Respiratory Care
Orlando, Florida Dec. 2-5, 1995**

WITH THE INFORMATION SERVICE CARD, YOU CAN STOP SEARCHING AND START BUYING.

Get the facts on all the
products and services advertised in
this issue easily and quickly.
The computerized Information
Service Card does it all.
Simply fill in your name and
address, check
the appropriate boxes, and
mail or fax it.

For your convenience, and direct access, the advertisers in this issue and their phone numbers are listed below. Please use this directory for requesting written material or for any question you may have.

HELP LINES

AARC Information	214-243-2272
Armstrong Medical	800-323-4220
DHD Diemolding Healthcare Systems	800-847-8000
Dräger Critical Care Systems	703-817-0100
HealthScan Products Inc	800-962-1266
Impact Instrumentation	800-969-0750
Nellcor	800-NELLCOR
Newport Medical	800-451-3111
Puritan-Bennett Corp	800-255-6773
Respironics	800-345-6443
Sherwood Medical	800-325-7472

Get Him Off The Vent And Back Into Life.

.....

Newport's Wave VM200 ventilator is designed to quickly wean even the most difficult patients.

.....

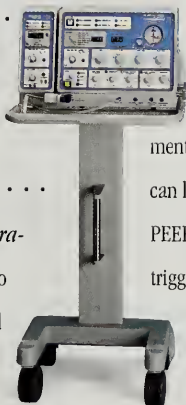
Effectively weaning your patients from mechanical ventilation is critical to returning them to productive, healthy lifestyles. Inefficient ventilation can not only delay a patient's recovery, but can increase hospital costs. The Wave VM200 provides the information and tools you need to make simple adjustments for superior patient weaning.

.....

Tools to Synchronize Patient and Ventilator

.....

For example, the *Peak Inspiratory Flow Monitor* enables you to assess the adequacy of mechanical flow versus patient flow demand and adjust accordingly.



Using the *Peak Expiratory Flow Monitor* can help you determine if a filter is clogged, assess a response to a bronchodilator and observe changes in compliance and resistance through

increased or decreased peak flows. By making appropriate adjustments on the ventilator you can lessen the chance for auto-PEEP and decrease work to trigger the ventilator.

Also facilitating recovery is our *Predictive Learning Logic*

software which automatically anticipates the natural lung movement and works with the patient to decrease breathing effort and reduce stress. And working in any ventilator mode, our unique *Bias Flow* decreases ventilator response time and stabilizes baseline pressure.

Call today for a complimentary demonstration of the Wave VM200, number one for patient weaning.

NMI
NEWPORT MEDICAL INSTRUMENTS, INC.

Post Office Box 2600, Newport Beach, CA 92658
800. 451. 3111 714. 642. 3910
Fax 714. 548. 3091

Please Visit Us At Booth #535, ALA/ATS

**NOW WITH
INTEGRATED ASTHMA
MANAGEMENT
ZONE SYSTEM**

PEAK MONITORING

Patented flow-sampling technology minimizes wear... maximizes accuracy and reproducibility

Sterilizable... dishwasher-safe

Meets NAEP Technical Standards

Three-Zone Management... made easy

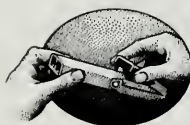
Easy-to-read scale — available in Full (60-810 L/min) and Low (50-390 L/min) ranges

Forget everything you know about peak flow meters.

We did. Keeping only the proven accuracy of our exclusive flow-sampling technology, we set out to build an entirely new kind of



The self-contained carrying case...



...turns into a handle...

peak flow meter. The result is Personal Best® — the revolutionary new meter that's changing the

shape of peak flow monitoring.

Wherever Personal Best goes — in the hospital, office, or home — it sets a new standard for convenience, compliance,

and confidence.



...makes using Personal Best child's play.

And Personal Best goes anywhere...easily. With a lightweight, self-contained, portable design that puts peak flow monitoring at your fingertips — or your patients' — all day long.

Plus a rugged, sterilizable construction that helps it stand up to the roughest use day after day.

Personal Best encourages compliance with its comfortable oval mouthpiece and easy-to-use built-in handle. And it improves patient communication with its high-legibility scale and NAEP-endorsed Three-Zone Management System.



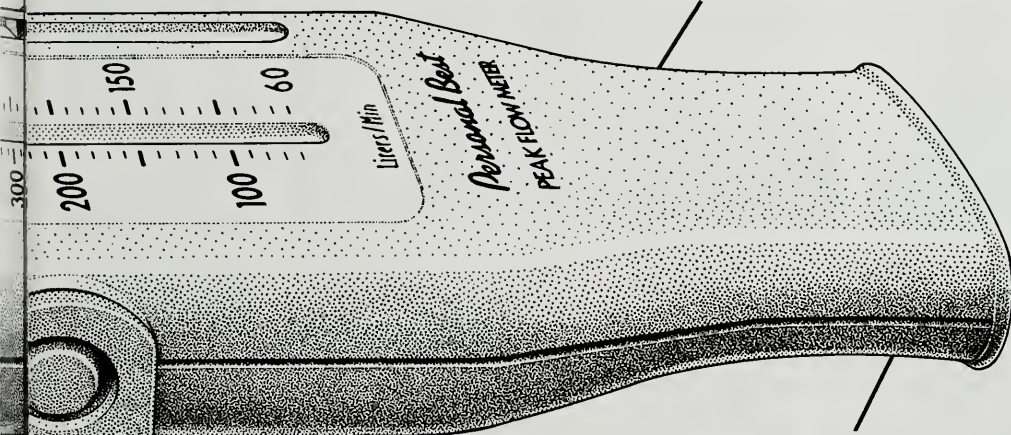
Personal Best travels light & inconspicuously — goes wherever you and your patients go.

Comprehensive asthma education program.



FLOW REDEFINED.

High-impact ABS plastic construction, surgical stainless steel moving parts — for reliable, cost-effective performance



Foldout handle keeps hands away from airstream — ensures proper technique for accurate readings

Most important, Personal Best provides accurate, instant airflow measurements that may be used by healthcare professionals to guide patient screening, emergency assessment, discharge instruction, medication adjustment, and home monitoring.

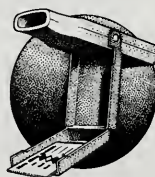


Sterilizable construction and disposable mouthpieces make multi-patient screening a snap.



Better fit.
Better comfort.
Better seal.

Unique oval mouthpiece — easier and more comfortable for patients to use



Instruction card fits inside handle.

Get your hands on Personal Best, and experience the difference for yourself. Call **1-800-962-1266** toll-free. Or drop this coupon in the mail.

RC 05/95

Personal Best® Peak Flow Meter

Redefining peak flow monitoring.

I want to experience the Personal Best difference first-hand.

Name _____
 Institution _____
 Address _____
 City _____ State _____ ZIP _____

Telephone _____ © 1995 HealthScan Products Inc. PA755002-2
HealthScan Products Inc. • 908 Pompton Avenue • Cedar Grove, NJ 07009-1292 USA
 1-800-962-1266 FAX (201) 239-0831



AMERICAN ASSOCIATION FOR RESPIRATORY CARE
11030 Ables Lane, Dallas, TX 75229 • 214/243-2272, Fax 214/484-2720

Dear Reader:

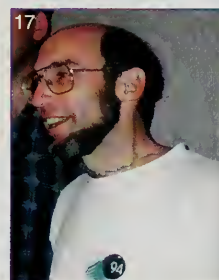
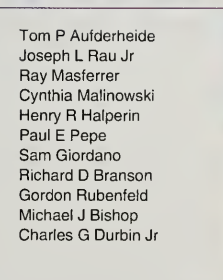
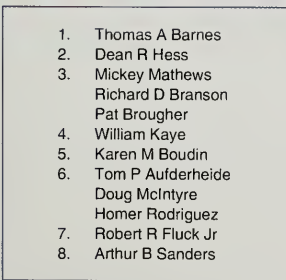
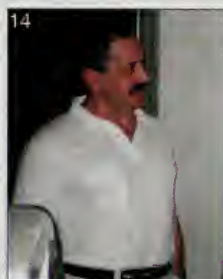
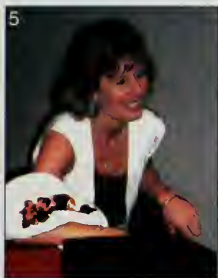
Beginning in 1981, and every year thereafter, the AARC's science journal, RESPIRATORY CARE, has held a state-of-the-art conference for the purpose of generating timely special issues on topics of interest to the AARC membership in particular, and to the entire pulmonary community in general.* The Conference proceedings issues are probably the most important ones we publish all year. They concentrate unique information in one place, some of which has never before been presented. The April and May 1995 issues contain the Proceedings from the 1994 Journal Conference "Resuscitation in Acute Care Hospitals." We are proud to bring you these papers and discussions, designed for you to use in your daily practice.

Presentation and publication of a conference like this requires considerable planning. The AARC and the Journal thank the speakers, the Editorial Board, Conference Co-Chairmen Tom Barnes and Charlie Durbin, Editor Pat Brougher, and Associate Editor Kaye Weber for planning, writing, and editing these special issues and for participating in the Conference and sharing this important information with us. Last, but certainly not least, we are extremely grateful to Allen & Hanburys, Division of Glaxo Inc, for their generous grant to the Association, which made the presentation of the Conference and publication of its Proceedings possible. Allen & Hanburys has supported the last seven Journal Conferences, and we are enormously indebted to them.

Sincerely,

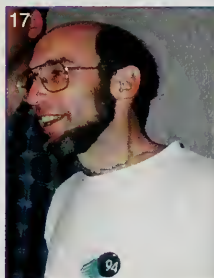
Managing Editor, RESPIRATORY CARE

- *1982—Complications of Respiratory Therapy (April, Volume 27, Number 4)
- 1983—The Management of Acute Respiratory Failure (May, Volume 28, Number 5)
- 1984—Perioperative Respiratory Care (May and June, Volume 29, Numbers 5 and 6)
- 1985—Monitoring of Critically Ill Patients (June and July, Volume 30, Numbers 6 and 7)
- 1986—Neonatal Respiratory Care (June and July, Volume 31, Numbers 6 and 7)
- 1987—Mechanical Ventilation (June and July, Volume 32, Numbers 6 and 7)
- 1988—PEEP (June and July, Volume 33, Numbers 6 and 7)
- 1989—Pulmonary Function Testing (June and July, Volume 34, Numbers 5 and 7)
- 1990—Noninvasive Monitoring in Respiratory Care (June and July, Volume 35, Numbers 6 and 7)
- 1991—Respiratory Care of Infants and Children (June and July, Volume 36, Numbers 5 and 7)
- 1991—Consensus Conference on Aerosol Delivery (September, Volume 36, Number 9)
- 1992—Emergency Respiratory Care (June and July, Volume 37, Numbers 6 and 7)
- 1992—Consensus Conference on the Essentials of Mechanical Ventilators (September, Volume 37, Number 9)
- 1993—Oxygenation in the Critically Ill Patient (June and July, Volume 38, Numbers 6 and 7)
- 1994—Controversies in Home Respiratory Care (April and May, Volume 39, Numbers 4 and 5)



1. Thomas A Barnes
2. Dean R Hess
3. Mickey Mathews
4. Richard D Branson
5. Pat Brougher
6. William Kaye
7. Karen M Boudin
8. Tom P Aufderheide
9. Doug McIntyre
10. Homer Rodriguez
11. Robert R Fluck Jr
12. Arthur B Sanders

9. Tom P Aufderheide
10. Joseph L Rau Jr
11. Ray Masferrer
12. Cynthia Malinowski
13. Henry R Halperin
14. Paul E Pepe
15. Sam Giordano
16. Richard D Branson
17. Gordon Rubinfeld
18. Michael J Bishop
19. Charles G Durbin Jr



RESUSCITATION IN ACUTE CARE HOSPITALS

Part II

A Special Issue

based on a conference
held October 21-23, 1994
in Cancun, Mexico

sponsored by
The American Association for Respiratory Care
and its science journal **RESPIRATORY CARE**
with the aid of an education grant
from Allen & Hanburys, Div of Glaxo Inc

Chairmen & Guest Editors:

Thomas A Barnes EdD RRT and Charles G Durbin Jr MD

Speakers:

Tom P Aufderheide MD
Thomas A Barnes MD
Michael J Bishop MD
Karen M Boudin MA RRT
Richard D Branson RRT
Charles G Durbin Jr MD
Robert R Fluck Jr MEd RRT
Henry R Halperin MD

Robin Hamill MD
Dean R Hess PhD RRT
William Kaye MD
Cynthia Malinowski MA RRT
Paul E Pepe MD
Joseph L Rau Jr PhD RRT
Gordon D Rubenfeld MD
Arthur B Sanders MD

Discussants:

The chairmen, the speakers, Sam P Giordano MBA RRT,
Ray Masferrer RRT, Paul J Mathews MPA RRT, Mickey
Mathews RN, David J Durand MD, Doug McIntyre RRT,
Homer L Rodriguez RRT

Ventilatory Support during Cardiopulmonary Resuscitation

Richard D Branson RRT and Jay A Johannigman MD

- I. INTRODUCTION**
 - A. Standards for Ventilation & Devices**
 - B. Lung Compliance following Cardiac Arrest**
 - C. Gastric Insufflation**
- II. EMERGENCY VENTILATION: ISSUES, TECHNIQUES & DEVICES**
 - A. Expired Air Resuscitation (EAR)**
 - 1. Issues Affecting EAR**
 - 2. Mouth-to-Mouth Resuscitation**
 - 3. Barrier Devices**
 - 4. Mouth-to-Mask Ventilation**
 - B. Bag-Valve Resuscitator Devices**
 - 1. Description**
 - 2. Assessment and Controversies**
 - C. Oxygen-Powered Breathing Devices**
 - 1. Description**
 - 2. Assessment & Limitations**
 - D. Ventilators for Resuscitation**
 - 1. Description**
 - 2. Characteristics**
 - 3. Assessment and Comparisons**
- III. FUTURE RESEARCH**

INTRODUCTION

Ventilatory support during cardiopulmonary resuscitation (CPR) can be accomplished by a number of methods and devices. These extend across a gamut from expired air resuscitation (EAR), including mouth-to-mouth and mouth-to-mask ventilation, to the use of ventilators includ-

ing ventilator-to-mask and ventilator-to-artificial-airway techniques. Appropriate application of these techniques depends on the clinical situation, rescuer training, and availability of equipment.

In this paper, we discuss the proposed standards for emergency ventilatory support, the advantages and disadvantages of techniques and devices used, and current controversies surrounding this topic.

Standards for Ventilation & Devices

Several agencies have published recommendations for the use and evaluation of emergency ventilation devices. These include the American Heart Association (AHA), the Emergency Care Research Institute (ECRI), the American Society of Testing Materials (ASTM), and the International Standards Organization (ISO).¹⁻⁹ Of these, only the AHA provides standards for the level and timing of ventilation as well as for device characteristics.¹ The other agencies sug-

Mr Branson and Dr Johannigman are Assistant Professors of Surgery associated with the Division of Trauma and Critical Care, Department of Surgery, University of Cincinnati Medical Center, Cincinnati, Ohio.

A version of this paper was presented by Mr Branson during the RESPIRATORY CARE Journal Conference "Resuscitation in Acute Care Hospitals" held in Cancun, Mexico, October 21-23, 1994.

The authors have no financial interest in any of the products mentioned in the text.

Reprints: Richard D Branson, Dept of Surgery, Univ of Cincinnati Medical Center, 231 Bethesda Ave, Cincinnati, Ohio.

gest standards for rate and volume but are more focused on the testing of devices.

Current AHA recommendations for adults include a frequency (f) of 10-12 breaths/min, a tidal volume (V_T) of 0.8-1.2 L, and an inspiratory time of 1.5-2.0 seconds. This corresponds to an inspiratory flow of 30-40 L/min.¹ The use of inspiratory times longer and flows lower than those usually encountered during spontaneous breathing are recommended to prevent gastric insufflation.^{10,11} ISO and ASTM standards suggest a V_T of 0.6 L and f of 12 breaths/min.

According to the new AHA Guidelines¹ all devices used for ventilation can be classified in the same manner as other resuscitation adjuncts, maneuvers, and drugs. The AHA classification, which is referred to during device descriptions, encompasses

Class I. A therapeutic option that is usually indicated, always acceptable, and considered useful and effective.

Class II. A therapeutic option that is acceptable, but of uncertain efficacy, and may be controversial.

Class IIa. A therapeutic option for which the weight of evidence is in favor of its usefulness and efficacy.

Class IIb. A therapeutic option that is not well established by evidence but may be helpful and probably is not harmful.

Class III. A therapeutic option that is inappropriate, is without scientific supporting data, and may be harmful.

Lung Compliance following Cardiac Arrest

The changes in compliance and resistance that follow cardiopulmonary arrest have been attributed to pulmonary aspiration, pulmonary venous congestion, effects of chest compressions, and pulmonary embolism.¹²⁻¹⁴ These studies typically compare blood gas values to postmortem examinations of the lungs. A common finding in these reports is pulmonary edema with wet, heavy lungs.¹²⁻¹⁴ Fillmore et al¹² made comparisons of arterial blood gas values (ABGs) to postmortem lung weights and found the lightest lungs were associated with the best ABGs.

Ornato et al¹⁵ measured ventilation during CPR and estimated pulmonary compliance. The investigators in this study used a pressure-limited ventilator to provide mechanical ventilation during CPR via an endotracheal tube. V_T was measured with a Wright respirometer. Ornato and colleagues constructed compliance curves using the ventilator connected to a test lung and used regression analysis from these data to estimate patient lung compliance from peak inspiratory pressure (PIP) and V_T measurements.

This technique more nearly estimates dynamic lung compliance and suffers from other methodologic problems, including the accuracy of volume measurement, the ability of the test lung to simulate the clinical situation, interaction between the pressure-limited ventilator and chest

compressions, and ventilator operation. According to Ornato et al¹⁵ lung compliance during cardiac arrest is 0.022 L/cm H₂O or about one fourth of normal lung compliance. This value is often used in laboratory experiments to compare emergency ventilation techniques and devices.

In 1994, our group evaluated lung compliance in patients in the emergency department following cardiac arrest.¹⁶ Twenty-five patients in cardiac arrest were studied immediately following discontinuation of CPR. Prior to measurements, the endotracheal tube cuff was inflated to assure a proper seal and appropriate position of the tube was confirmed by auscultation and/or chest radiograph. A super syringe was used to inflate the lungs in 0.25-L increments from 0.25 L to 2.0 L. An inspiratory pause of 2.0 seconds was used, and flow and pressure were measured at the proximal airway with a pneumotachograph and an airway pressure transducer. All raw data were saved to the hard drive of a computer and used to construct compliance curves.

Data from the study are shown in Figure 1. The mean compliance was 0.051 L/cm H₂O, a value more than twice that reported by Ornato et al.¹⁵ At low V_T s (< 0.5 L), compliance was 0.04 L/cm H₂O, suggesting that functional residual capacity was reduced. At AHA-recommended volumes (0.8-1.2 L), mean lung compliance was 0.05 L/cm H₂O. These findings have implications for future emergency ventilation research and interpretation of previous studies. The use of longer inspiratory times and lower flows during CPR is, in part, related to the suspected low lung compliance (0.02 L/cm H₂O). Perhaps ventilation strategies should be modified based on findings that lung compliance is twice that previously reported.

Another variable affecting delivered V_T is esophageal opening pressure. As airway pressure is increased to deliver volume in the face of increased pulmonary impedance, gastric insufflation results. Based on work by Ruben et al,¹⁰ esophageal opening pressure is around 20 cm H₂O.

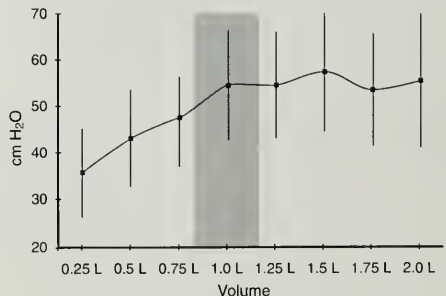


Fig. 1. Mean pulmonary compliance of 25 patients following cardiac arrest. Gray area delineates AHA-recommended volume.

Gastric Insufflation

Gastric insufflation with the potential sequela of pulmonary aspiration is a problem whenever ventilation via an unprotected airway is attempted. An effective strategy for limiting gastric insufflation includes using less forceful inspirations, longer inspiratory times, and smaller tidal volumes.¹⁷

Another technique to prevent gastric insufflation is the use of cricoid pressure, often referred to as the Sellick maneuver,¹⁸ and described by Sellick in 1961 as a method to prevent regurgitation during induction of anesthesia and endotracheal intubation. By pushing down on the cricoid membrane, the rescuer collapses the esophagus against the cervical vertebrae. This is illustrated by photographs from Sellick's original paper shown in Figure 2.

Use of cricoid pressure during CPR appears attractive. However, AHA recommends that cricoid pressure be used only by health care professionals during two-rescuer CPR. Even in this situation, cricoid pressure can only be applied by a third rescuer. During mouth-to-mouth or mouth-to-mask ventilation, the rescuer providing ventilation needs both hands for airway control, and the rescuer performing compressions obviously 'has his hands full.' The Sellick maneuver is relatively simple and could be accomplished by an untrained assistant under direction of a rescuer. The chance of having three rescuers during CPR, however, seems remote.

In children, cricoid pressure has been shown to prevent gastric insufflation during mask ventilation up to a PIP of 40 cm H₂O.¹⁹ Vanner et al found that a cricoid force of 40 N (ie, newtons) increased esophageal opening pressure to a mean of 38 torr in anesthetized adults.²⁰

Cricoid pressure is a fairly simple technique but is not without complications. If excessive pressure is applied, the trachea can also be collapsed, causing complete airway obstruction. There are also reports of gastric rupture occurring when the patient regurgitates during application of cricoid

pressure.²¹ Gastric rupture typically involves the lesser curvature of the stomach and is related to elevated pressures created when the esophagus is effectively closed.²²

EMERGENCY VENTILATION: ISSUES, TECHNIQUES, & DEVICES

Expired Air Resuscitation (EAR)

Issues Affecting EAR

EAR includes mouth-to-mouth, mouth-to-mask, and mouth-to-nose ventilation. Mouth-to-mouth ventilation is probably the oldest method of EAR and is mentioned in the Bible.²³

Disadvantages of EAR include low delivered oxygen concentration (F_{DO_2}), gastric insufflation, unpleasantness of the task, fear of disease transmission, and actual disease transmission.

As previously described, during EAR a V_T of 0.8-1.2 L should be delivered over a period of 1.5-2.0 s, 10-12 times/min. This results in a minute ventilation ($\dot{V}_E$) of 8.0-14.6 L/min and an inspiratory-to-expiratory-time ratio of 1:1.5 to 1:2.

F_{DO_2} during EAR. During EAR, F_{DO_2} is 0.16-0.18, allowing for an alveolar oxygen tension of 52 to 76 torr, assuming a barometric pressure of 747 torr and P_{aCO_2} of 40 torr. This range of P_{aO_2} is associated with an oxygen saturation of about 90%. Hypoxemia during cardiac arrest is much more likely to be caused by low cardiac output and ventilation-perfusion abnormalities than by low F_{DO_2} .¹ During mouth-to-mouth ventilation, F_{DO_2} can be increased by increasing inspired oxygen concentration (F_{IO_2}) of the rescuer. Hess et al²⁴ evaluated the change in F_{DO_2} when the rescuer was wearing a nasal cannula with oxygen flows of 6 L/min and 10 L/min. They found mean (SD) values for F_{DO_2} of 0.16(0.01) during EAR, 0.27(0.03) at an oxygen flow of 6 L/min, and (0.31)(0.05) at an oxygen flow of 10

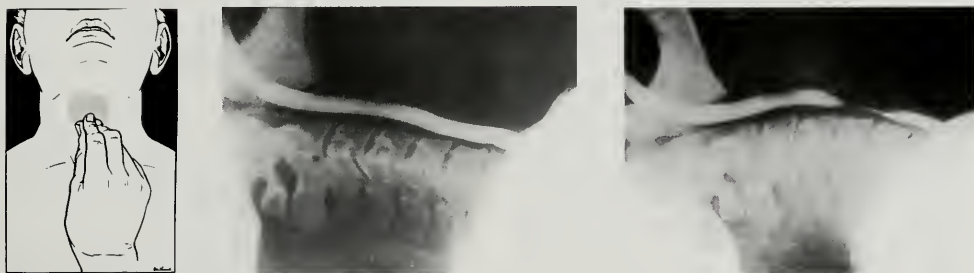


Fig. 2. (A.) Diagram showing the Sellick maneuver; (B.) radiograph of neck showing esophageal lumen filled by contrast medium; (C.) radiograph showing lumen with cricoid pressure applied. (From Reference 18, with permission)

L/min. Hess and colleagues suggested that in the absence of other devices, oxygen breathing by the rescuer is a viable method of delivering adequate V_T and elevated F_{DO_2} .

In a lung model study, Rottenberg et al.²⁵ found mean (SD) values for F_{DO_2} of 0.18 (0.05) during EAR and 0.32 (0.03) when the rescuer was breathing oxygen via a nasal cannula at 10 L/min. They also evaluated the role of oral inspiration of oxygen via the supply tubing at 15 L/min. With this technique, the rescuer places the connecting tubing between his lips and inspires just prior to ventilating the mannequin. Lastly, rescuers breathed air from a manually triggered demand valve. The demand valve was placed between the rescuer's lips and the manual trigger activated. In this instance, the rescuers were essentially ventilating themselves with 100% oxygen via the demand valve prior to ventilating the mannequin. Breathing oxygen from the supply tubing allowed an F_{DO_2} of 0.37 (0.10) and inspiring from the demand valve provided an F_{DO_2} of 0.78 (0.09). The authors suggested that patients at risk for cardiac arrest should keep oxygen on hand in the home to allow family members to use supplemental oxygen during resuscitative efforts. They likewise suggested placing oxygen in high risk areas, such as near swimming pools.

Both these studies make conclusions about the potential usefulness of increasing rescuer F_{IO_2} to increase F_{DO_2} . However, it seems likely that wherever oxygen is available, bag-valve-mask (BVM) ventilation is also probably available. In the study by Rottenberg et al.,²⁵ it is unclear why if the rescuer has a demand valve, it is not used to ventilate the patient. These data do prove, however, that F_{DO_2} can be increased by increasing rescuer F_{IO_2} .

Disease Transmission during EAR. Concern about disease transmission has become a major issue in the delivery of bystander and first responder CPR including mouth-to-mouth resuscitation.²⁶⁻³¹ Ornato et al.²⁶ found that 40% of BCLS instructors, including health care workers, public-service workers, and laypersons, would hesitate to perform mouth-to-mouth ventilation for fear of catching a disease.²⁶

Additional surveys have found that although many BCLS providers say they would perform mouth-to-mouth ventilation on a stranger (97%), only one half (44%) would perform CPR on a patient known to have or suspected of having AIDS.²⁷ Link et al.²⁸ found that 48% of medical residents employed in New York City hospitals reported a moderate-to-major concern about contracting AIDS from a patient. These studies do not address the possibility of contracting a disease following mouth-to-mouth resuscitation, but suggest that a major impediment to out-of-hospital CPR is fear of disease transmission.

The AHA has addressed the issue of infection risk during CPR.^{1,29} When a patient is known to be "high risk," recommendations regarding ventilation techniques in-

clude: (1) rescuers who have an infection that may be transmitted by blood or saliva or who believe that they have been exposed to such an infection should not perform mouth-to-mouth ventilation if other methods are available (eg, mouth-to-mask or BVM); (2) individuals have a duty to respond to the CPR needs of high-risk patients using a mouth-to-mask device of adequate design or a BVM device and should be trained in their use; and (3) early intubation should be encouraged when equipment and trained professionals are available.²⁹

The effect of these suggestions on bystander CPR has been debated in these pages.^{30,31} An understanding of the use of bystander CPR is required prior to embarking on this discussion. Bystander CPR is primarily performed by family members and public health-service personnel.¹ In the latter case, equipment designed to obviate the need for mouth-to-mouth contact is available. In the former, the caregiver is knowledgeable of the patient's health history and is quite frequently a relative. The issue then, may be more academic than practical.

There remains, however, a reluctance on the part of many people to perform mouth-to-mouth resuscitation. Although the risk of infection is small, there are reports of possible instances of infection following CPR, including infection with *Mycobacterium tuberculosis*,³² meningococcus,³³ herpes simplex,³⁴⁻³⁶ shigella,³⁷ and salmonella.³⁸

Health-care policy has salded to the confusion on disease transmission. Although the risk of infection is reported to be low and the risk of HIV transmission even smaller, recommendations are made that devices to prevent mouth-to-mouth contact be available in high-risk areas.^{29,39} The City of New York has even made the availability of infection control equipment for CPR in public places mandatory.⁴⁶

Debate about the true risk of infection from performing CPR may be futile. The unpleasantness of the task of mouth-to-mouth breathing (because of the presence of vomitus and blood, for example) and even the minute possibility of infection mandates that we develop cost-effective, readily available, safe, and efficacious alternatives to mouth-to-mouth breathing.

Mouth-to-Mouth Resuscitation

Description. Mouth-to-mouth ventilation is accomplished by the rescuer placing his or her mouth over the victim's mouth and exhaling while maintaining a patent airway. The rescuer should watch the rise and fall of the victim's chest, listen for air escaping during exhalation, and feel the exhaled air flow.

Mouth-to-mouth ventilation has been recommended for Basic Life Support (BLS) since 1974. The major advantages of mouth-to-mouth ventilation are availability, ease of use, universal application, large reservoir volume (the

delivered volume is limited only by the rescuers vital capacity, which is 3 to 4 times the necessary V_T , and the fact that no equipment is necessary.

Assessment. Numerous publications have shown that mouth-to-mouth ventilation is effective in providing AHA recommended V_T s, regardless of lung compliance.⁴¹⁻⁴⁶ Our group⁴⁶ found that mouth-to-mouth was superior to mouth-to-mask, BVM, and ventilator-to-mask ventilation with respect to delivered V_T (Fig. 3). However, mouth-to-mouth ventilation is also associated with the greatest amount of gastric insufflation.⁴⁶ The increase in gastric insufflation is due to the rescuer's ability to deliver a larger, more forceful volume when pulmonary impedance is increased.

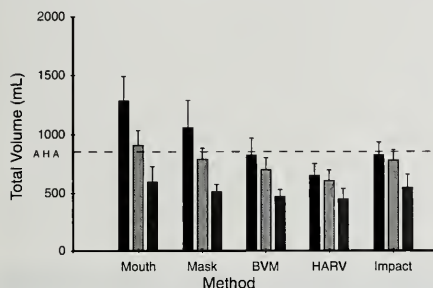


Fig. 3. Delivered tidal volume with 5 methods of ventilation. * = $p < 0.05$ vs other methods, with same compliance. ■ = 0.1 L/cm H_2O ; ▒ = 0.04 L/cm H_2O ; □ = 0.02 L/cm H_2O ; HARV = Hope Adult Resuscitation Ventilator (From Reference 42, with permission).

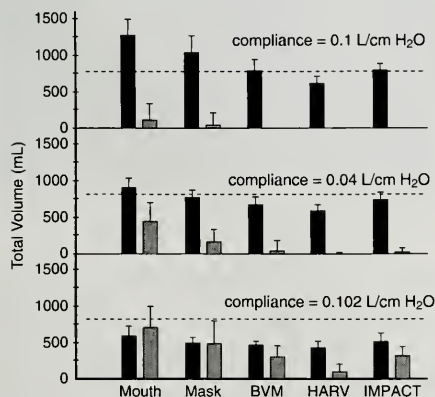


Fig. 4. Comparison of delivered V_T versus gastric insufflation. Dashed line represents AHA minimum volume of 800 mL. ■ = Tidal Volume; ▒ = gastric volume (From Reference 42, with permission).

Other data from our group suggest that with normal lung compliance, gastric insufflation during mouth-to-mouth ventilation is low (Fig. 4⁴⁶). This is in agreement with work by Melker and Banner.¹⁷

Barrier Devices

Description. A barrier device is a flexible sheet that typically contains a valve and/or filter that separates the rescuer from the patient (Fig. 5). Barrier devices are a disposable version of the "handkerchief" recommended by Waters as a protective device some 50 years ago.⁴⁷ Barrier devices are sometimes called face shields but should not be confused with face masks. An effective barrier device should have:

- Universal application—conforming to the anatomy of patients of all sizes and shapes.
- Be small, lightweight, and easy to carry.
- Have minimal air flow resistance.
- Prevent cross-infection (victim to rescuer and vice-versa).
- Resist tearing.

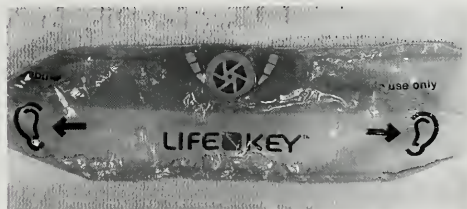


Fig. 5. Barrier device used for CPR.

Assessment. Few evaluations have been made on the safety and efficacy of barrier devices. Anecdotal reports^{48,49} of the ability of barrier devices to prevent movement of HIV-infected broth have been published. A mannequin study found that ventilation with a barrier device provided a level of ventilation similar to mouth-to-mouth ventilation without a barrier device.⁵⁰ In a 1989 *Health Devices* report,⁸ barrier devices were felt to be inferior to face masks with respect to creating an effective seal on the face. There is also some concern that barrier devices may slip if the patient's face is wet and that during prolonged resuscitation efforts, the plastic sheet may tear away from the central valve.⁵¹

Lightsey et al⁴⁹ evaluated the Kiss of Life barrier device in the laboratory using a mannequin and in the operating room with anesthetized patients. Two rescuers ventilated the mannequin using direct mouth-to-mouth ventilation, the Kiss of Life barrier device, the Microshield barrier device, the Laerdal pocket mask, a manual resuscitator, and a Brooks airway, for 3 minutes. The data, which are presented without standard deviations showed a mean volume of

1.0 L for mouth-to-mouth, 0.7 L for the Kiss of Life, 0.62 L for the Microshield, 0.47 L for the Laerdal pocket mask, 0.46 L for the manual resuscitator, and 0.2 L for the Brooks airway. In the operating room, 10 anesthetized patients were ventilated with 2 breaths using the Kiss of Life barrier device, 2 breaths via BVM, and 2 breaths with bag-valve-mask, following endotracheal intubation. Delivered volumes were 1.1 L, 0.7 L, and 1.5 L, respectively. This paper, one of the few evaluations performed, suffers greatly from methodologic and statistical errors. The results of this paper should be considered preliminary at best and suspect at worst. Additionally, the authors designed the Kiss of Life device and may have been biased in their evaluation. Westfall has criticized this paper for similar reasons.⁵²

More recently, Rossi et al⁵³ have compared mouth-to-mask, mouth-to-tube, and mouth-to-barrier device methods of ventilation in the laboratory. Using a motorized calibration syringe, they delivered a V_T of 1.0 L to a mannequin and test lung. Inspiratory and expiratory pressures and valve leakage were measured. Results from this study are shown in Table 1. Rossi et al⁵³ concluded that resistance to air flow was excessive in several of the devices tested. This has been confirmed by Simmons⁵⁴ in a recent abstract. He found that of three barrier devices (Kiss of Life, Microshield, and Res-Cue Key) only the Res-Cue Key met the ISO standard of < 5 cm H₂O back pressure at a flow of 50 L/min. In this report, the Microshield device created almost 17 cm H₂O back pressure at a flow of 50 L/min.

Table 1. Average Inspiratory and Expiratory Pressures and Valve Leakage of Expired-Air Resuscitation Devices*

Device	Inspiratory Pressure (cm H ₂ O)	Expiratory Pressure (cm H ₂ O)	Valve Leakage (mL)
Mask Instruments			
Air-Vita Bi-Protect	8.8	5.8	5.0
Laerdal Pocket Mask	5.8	10.0	<1.0
Dräger Hivita Mask E	2.4	6.7	27.0
Rescue-Med	9.5	3.5	5.0
Resuscitator	4.9	4.4	10.0
SealEasy Resus. Kit	1.5	1.8	2.0
Pharyngeal Tubes			
Dr Brook Airway	39.0	21.0	24.0
Dual-Aid	9.8	15.3	48.0
Goettlinger Tubus	8.8	10.4	Exp. Vol.
Lifeway	2.5	1.6	2.5
Sussex Valve Airway	3.7	3.6	<1.0
Foils			
Ambu Life-Key	1.8	undetectable	<1.0
Resusciface Shield	4.7	undetectable	undetectable

*From Reference 53, with permission.

Simmons and co-workers⁵⁵ also compared volume delivered to a mannequin using mouth-to-mouth and these

same three barrier devices. They reported a volume of 1.0 L using mouth-to-mouth, 0.25 L using the Kiss of Life, 0.75 L using the Microshield, and 0.64 L using the Res-Cue Key. The reasons for these disparities are unclear.

More research is necessary to determine the safety and efficacy of barrier devices. AHA considers barrier devices as Class IIb (acceptable, possibly helpful).¹

Mouth-to-Mask Ventilation

Description. Mouth-to-mask ventilation devices can be as simple as a face mask or can include a face mask, oxygen inlet, nonrebreathing valve (NRV), extension tube, filter, and mouthpiece (Fig. 6). Both devices can be used effectively, but the latter incorporates components that help protect the rescuer and patient from cross-contamination.

I believe that desirable characteristics of a mouth-to-mask device include a clear, soft mask capable of making an effective seal with the patient's face; an oxygen inlet nipple; an NRV that diverts expired flow away from the rescuer, low flow resistance, no back leak, and a filter with low flow resistance that is not adversely affected by humidity or vomitus. Dead space should be considered in pediatric applications. In adults, a dead space < 200 mL is acceptable.



Fig. 6. Mouth-to-mask resuscitation device with (right) and without (left) an oxygen inlet.

Assessment. Mouth-to-mask ventilation was shown to be an effective method of ventilation as early as 1954 by Elam, Brown, and Elder.⁵⁶ Safar described the first mouth-to-mask device (Laerdal pocket mask) in 1958⁵⁷ and the role of oxygen supplementation in 1974.⁵⁸ For more than a decade, the technique of mouth-to-mask ventilation has been recommended by AHA as the method of choice prior to endotracheal intubation unless a rescuer with experience with BVM is present. Despite the use of the mask, proximity of the rescuer to the victim continues to be the major

detriment to widespread use of mouth-to-mask ventilation.

Mouth-to-mask ventilation has been shown to be superior to BVM ventilation in a number of studies.^{41-46,59-62} Harrison and colleagues⁴¹ found that unskilled rescuers delivered a mean volume of 942 mL to a mannequin using mouth-to-mask and only 495 mL with BVM ventilation. In a small group of skilled rescuers, ($n = 4$) volumes with mouth-to-mask averaged 1193 mL compared to only 509 mL with BVM ventilation. The authors recommended that mouth-to-mask ventilation replace the bag-valve-mask system in the initial management of respiratory arrest.

Hess and Baran,⁴² using a mannequin and test lung, found higher volumes were provided with mouth-to-mask compared to BVM ventilation by one rescuer. In this study, mean delivered volume with mouth-to-mouth was 0.73 L, mouth-to-mask was 0.6 L, and BVM ventilation was 0.3 L. When two people used the BVM device, one to hold the mask seal and one to squeeze the bag, delivered volumes increased to 0.58 L. It is interesting that the authors found no difference in performance of any of these techniques related to operator experience. This is contrary to the AHA statement concerning the use of BVM ventilation if an experienced user is present. Hess and Baran's work suggests that even with experience, BVM ventilation by a single rescuer is inadequate.

Seidelin et al⁴⁵ found that medical students provided the largest tidal volumes using mouth-to-mouth ventilation (2.9 L) and the smallest volumes using the Brook airway (0.44 L). In this study, mouth-to-mask ventilation provided 2.6 L volumes and single-rescuer BVM 0.7 L. The reason for the large tidal volumes is related to the study design. The students were asked to provide "5 maximal inspirations" within a 30-second period. These authors recommended that ventilation be provided by mouth-to-mask until a third rescuer arrives to allow two-person BVM ventilation.

Our group⁴⁶ found that mouth-to-mask ventilation consistently provided higher tidal volumes than BVM or ventilator-to-mask ventilation. That study used a mannequin that required active alignment of airway structures (head-tilt, chin lift) connected to a test lung with a variable compliance and resistance. Additionally, a simulated esophagus and stomach (Fig. 7) with an esophageal opening pressure of 20 cm H₂O was placed in line with the trachea. At normal compliance, 0.1 L/cm H₂O, we found that mouth-to-mouth, mouth-to-mask, BVM, and ventilator-to-mask ventilation provided adequate tidal volumes with little or no gastric insufflation (Fig. 4). At a compliance of 0.04 L/cm H₂O, which is near the value measured clinically, we found that mouth-to-mouth ventilation provided the largest tidal volumes but also produced gastric insufflation equivalent to 50% of the tidal volume. Mouth-to-mask ventilation produced similar tidal volumes but with approximately one third the gastric insufflation of mouth-to-mouth

ventilation. We made a further plea for early use of mouth-to-mask during single-rescuer CPR.⁴⁶

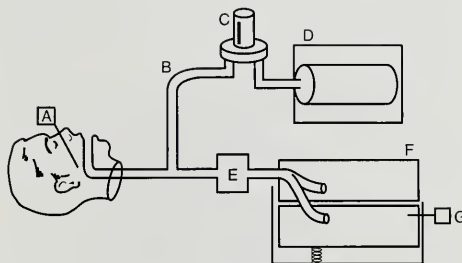


Fig. 7. Model for determining efficacy of prehospital ventilatory techniques. A = proximal airway pressure; B = 20-cm Penrose drain; C = water-column PEEP valve; D = rolling-seal spirometer; E = pneumotachograph; F = test lungs; G = distal airway pressure. (From Reference 42, with permission.)

Studies by Sainsbury et al⁴¹ and Lawrence and Sivanewaran⁴³ have also demonstrated the superiority of mouth-to-mask compared to BVM ventilation. Lawrence and Sivanewaran found that of 100 volunteers, only 3% provided a $V_T > 800$ mL with BVM ventilation compared to 54% when mouth-to-mask was used. When a period of training was completed, 97% using mouth-to-mouth ventilation provided a volume > 800 mL while the percentage of volunteers providing adequate ventilation with BVM was unchanged (3%).⁴³ Sainsbury et al⁴¹ studied 100 members of the nursing staff and found that 88% were capable of providing a minimum $\dot{V}_E$ of 6 L/min using mouth-to-mouth ventilation. In that same group, 66% could provide the pre-selected minimum $\dot{V}_E$ with mouth-to-mask, but only 25% were able to meet that requirement using BVM ventilation.

More recently, Thomas and colleagues⁵⁹ have evaluated mouth-to-mask ventilation using a mannequin and in anesthetized patients.^{60,62} These authors first placed a filter in the mouth-to-mask breathing device and determined its effect on rescuer performance.⁶² After determining that delivered volumes were unaffected and that the filter prevented transmission of bacteria, they proceeded to a human evaluation. In the follow-up study, the authors had anesthesia residents provide mouth-to-mask or BVM ventilation, both with an oxygen flow of 15 L/min, to 30 subjects (ASA Class I or II) requiring general anesthesia. Residents were given "brief tuition" by the investigators and used a Laerdal pocket mask or Laerdal silicone resuscitator for ventilation. During a 4-minute period, airway pressure, carbon dioxide concentration, and oxygen concentrations were measured. The authors found that BVM ventilation provided a mean

F_{DO_2} of 0.95 and mean delivered carbon dioxide (F_{DCO_2}) of 0.05. Mouth-to-mask ventilation yielded a mean F_{DO_2} of 0.54 and mean F_{PCO_2} of 0.14. Despite the higher F_{PCO_2} , mean expired CO_2 was equivalent between the groups, suggesting no adverse effect on CO_2 removal. Arterial oxygen saturation as measured by pulse oximetry was also equal between the two ventilation methods.

An interesting aside to this study⁶² is that during BVM ventilation a frequency of 18 breaths/min was provided whereas only 12 breaths/min was used during mouth-to-mask. Airway pressures were lower with BVM (17 cm H_2O) compared to mouth-to-mask ventilation (21 cm H_2O). These facts, when considered in light of the similar expired CO_2 data, suggest that V_T was increased with mouth-to-mask ventilation. The authors reported the occurrence of gastric insufflation in 4 patients (2 in each group). In each case, the authors observed difficulty in maintaining an open airway and a rise in airway pressures (mean 26 cm H_2O , range 20-28 cm H_2O). They also observed that cricoid pressure prevented further gastric insufflation. In agreement with the work of our group,⁴⁶ the authors found that gastric insufflation was greater with mouth-to-mouth than with BVM ventilation.

In a third study, Thomas and Weber⁵⁹ suggested using mouth-to-mask ventilation during two-rescuer CPR with an alternate strategy. Rather than one rescuer providing ventilation while the other performed compressions, they suggested that one rescuer hold the mask and provide airway control (head-tilt, chin lift), while the second rescuer provided both ventilation and compression. During conventional two-rescuer CPR, V_T was lower and respiratory rate was higher than during modified CPR. Table 2 shows the results of the study.

Table 2. Efficacy of Ventilation Provided to a Mannequin Using Standard CPR and Modified CPR.

Variable	Standard CPR*	Modified CPR
Frequency (breaths/min)	15 (2)	11 (2)
Tidal volume (mL)	640 (230)	1110 (166)
Minute volume (L)	9.7 (3.8)	12.6 (2.6)
Compression rate (breaths/min)	75 (10)	57 (11)

*Values are mean (SD).

From Reference 59, with permission.

Although the compression rate was slowed during modified CPR, the delivered V_T was significantly improved. The authors postulated that reducing the ventilation-compression ratio from 1:5 to 2:15 would increase the number of compressions while maintaining gas exchange, by virtue of the larger V_T . This method of BLS-CPR deserves further attention.

One of the drawbacks of mouth-to-mask ventilation is

the decreased F_{DO_2} . Safar demonstrated an F_{DO_2} of 0.54 using mouth-to-mask ventilation at a V_T of 1.0 L, frequency of 12 breaths/min, and oxygen flow of 15 L/min.⁵⁸ In an effort to increase F_{DO_2} , we⁶³ recommended placing the oxygen inlet valve of the mouth-to-mask device above the non-rebreathing valve and allowing the rescuer to inspire from the continuous oxygen flow during patient expiration. This so called inhalation technique was compared to conventional mouth-to-mask ventilation in a mannequin and lung model to determine the effect on F_{DO_2} . Oxygen flows of 5 L/min, 10 L/min, and 15 L/min were used. Figure 8 demonstrates the increase in F_{DO_2} seen with the inhalation technique. It is interesting that at a flow of 15 L/min, mean F_{DO_2} was 0.43, not far from that originally reported by Safar.⁵⁸ Using the inhalation technique, F_{DO_2} was increased to a mean of 0.71. We also noted that increasing oxygen flow was associated with increased delivered V_T . This is particularly evident at high oxygen flows, where rescuer expiratory effort is supplemented by the oxygen flow. At an oxygen flow of 15 L/min and inspiratory time of 1.5 seconds, an additional 375 mL of oxygen (250 mL/s) is added to the rescuer's effort. We cited previous work demonstrating the superiority of mouth-to-mask ventilation and suggested that oxygen supplementation using the inhalation technique might represent the ventilatory method of choice in early CPR prior to endotracheal intubation.

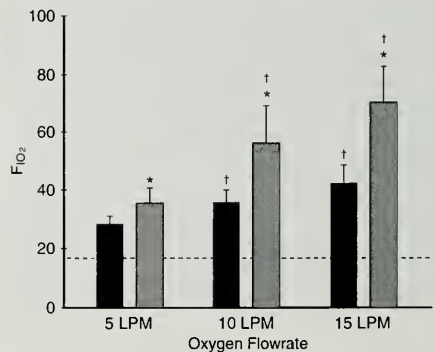


Fig. 8. Oxygen concentration at different oxygen flows, using standard and inhalation technique. ■ = standard; ▨ = inhalation; (---) = F_{IO_2} without enrichment; † = $p < 0.05$ vs previous flowrate/same technique; * = $p < 0.05$ vs standard technique. (From Reference 63, with permission.)

Stahl et al⁶⁴ studied five mouth-to-mask devices and evaluated the F_{DO_2} delivered to a test lung at two combinations of frequency and V_T (500 mL $\times$ 20 breaths/min and 90 mL $\times$ 12 breaths/min). A ventilator was used to keep rate and volume constant while oxygen flow to the mouth-

to-mask device was varied from 2 L/min to 14 L/min in 2-L increments. The devices performed similarly, with the limiting factor being dead-space volume of the mask. As dead-space volume increased so did F_{DO_2} . The device with the largest dead-space (300 mL) provided an F_{DO_2} of 0.58 at a V_T of 900 mL and rate of 12 breaths/min, and F_{DO_2} of 0.72 at a V_T of 500 mL and rate of 20 breaths/min (oxygen flow = 14 L/min). Whereas the smallest mask (dead space = 225 mL) provided an F_{DO_2} of 0.47 and 0.55. The authors recommended that during mouth-to-mask ventilation high oxygen flows (14 L/min) and slow inspirations be used.

Thomas et al⁶⁵ studied the effects of increasing oxygen flow to the Laerdal pocket mask. Using a recording mannequin, the authors had 24 volunteers provide mouth-to-mask ventilation for 90 seconds, with oxygen flows of 5 L/min, 10 L/min, 15 L/min, and 20 L/min. Similar to previous work, Thomas and colleagues found that, as oxygen flow increased inspired CO_2 decreased and V_T was enhanced. The authors suggested that a flow of 20 L/min is preferable if mouth-to-mask ventilation is used.

Palmisano and colleagues⁶⁶ evaluated the effect of supplementary oxygen flow on mouth-to-mask ventilation in a pediatric model. They demonstrated an F_{DO_2} of 0.50 with an oxygen flow of 5 L/min when V_T was 100 mL and frequency was 20 breaths/min. Increasing oxygen flow to 15 L/min served to increase F_{DO_2} to 0.60 but also increased delivered volume to 221 mL, a 121% increase. The authors also pointed out the adverse effect of introducing oxygen below the NRV. In this case, high flows interfere with function of the NRV.

The issue of increasing F_{DO_2} during mouth-to-mask ventilation appears to have been adequately addressed. Use of the inhalation technique may allow higher F_{DO_2} with lower oxygen flows, thus conserving oxygen supply. The more compelling question is related to the actual F_{DO_2} required during CPR.

Hess et al⁶⁷ compared mouth-to-mask devices and found considerable variability in device performance. In this study, 14 respiratory care practitioners ventilated a mannequin using mouth-to-mouth ventilation and 8 mouth-to-mask devices. They found the largest volumes were delivered with mouth-to-mouth ventilation (1.0 L) and volumes with the mouth-to-mask devices varied from 0.5 L to 0.8 L (Fig. 9). Hess and colleagues⁶⁷ attributed differences in device performance to design characteristics. They suggest specifically that differences are due to (1) ability of the mask to fit the face and achieve a seal; (2) resistance to flow through the NRV; and (3) size and shape of the mask, which either aids or hinders the rescuer's grip.

Mouth-to-mask ventilation combined with supplemental oxygen is arguably the safest, most effective method of ventilatory support in the intubated patient when one rescuer is present. Resistance to use of mouth-to-mask devices

may be related to rescuer proximity to the patient, with the attendant fears of disease transmission and the unpleasantness of the task. Mouth-to-mask ventilation may also appear unsophisticated when compared to BVM ventilation. Another issue may be related to a hindering of communication of the rescuer performing mouth-to-mask ventilation.

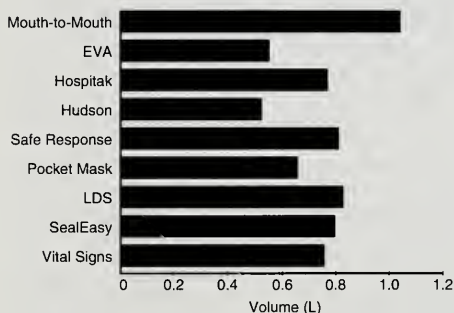


Fig. 9. Mean volumes delivered by mouth-to-mouth ventilation and mouth-to-mask ventilation with the devices evaluated. (From Reference 67, with permission)

Bag-Valve Resuscitator Devices

Description

Bag-valve resuscitators consist of a self-inflating bag, oxygen reservoir, and NRV (Fig. 10). The operator ventilates the patient by squeezing the self-inflating bag, which forces air into the NRV and to the patient. The self-inflating bag is typically made of a resilient material such as rubber, silicone, or polyvinylchloride. Most self-inflating bags have a volume of about 2.0 L for adults. When the operator releases the bag, it returns to its resting, inflated position. During re-expansion, the bag fills with room air via a one-way valve on the rear of the bag or with oxygen from an oxygen reservoir. Oxygen reservoirs usually are classified as either tube or bag reservoirs, based on their construction

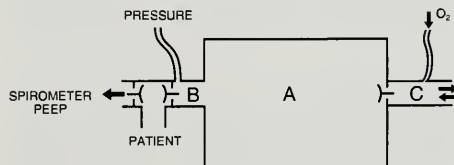


Fig. 10. Schema of a manual resuscitator. A. Self-inflating bag. B. Nonbreathing valve. C. Oxygen reservoir. (From Reference 68, with permission.)

and appearance. During the exhalation phase, the patient's expired air is directed away from the self-inflating bag to the environment by the NRV. NRVs are of several designs, including duck-bill valves, spring-disk valves, spring-ball valves, diaphragm valves, and leaf valves.

An ideal self-inflating resuscitator has the following characteristics:⁶⁸

- Bag volume should be twice the volume to be delivered and construction should allow the operator to deliver 800 mL while squeezing the bag with one hand.
- An F_{DO_2} of 0.85-1.0 should be available for CPR. The oxygen reservoir should be lightweight and unobtrusive.
- The construction of the bag should allow rapid refill so faster respiratory rates can be provided as required. The bag should not require gas flow to inflate, and the bag material should allow the operator a 'feel' for patient impedance characteristics.
- The NRV should prevent any back leak of patient expired gases into the bag, have a low resistance to inspiratory and expiratory flow (< 5 cm H_2O pressure drop at 50 L/min), and possess a minimum dead space (< 30 mL for adults). The NRV should be transparent to allow detection of vomitus or other obstruction and should perform effectively at high oxygen flows.
- Adult resuscitators should not have a pressure relief valve. Pediatric devices should be equipped with a 35-40 cm H_2O pressure relief valve that may be overridden if required.
- The bag-valve device should allow attachment of a spirometer to measure expired volumes and a positive-end expiratory pressure (PEEP) valve, and be equipped with a tap for monitoring airway pressure with an aneroid manometer.
- The resuscitator bag should be lightweight, easily held in one hand, easy to disassemble, easy to clean, and impossible to reassemble improperly. The device should be rugged and should perform well under adverse conditions of temperature.

Assessment & Controversies

In 1992, Barnes reviewed the literature on this topic in this journal.⁶⁹ In this portion of the paper, we deal with the current literature and controversies concerning BVM ventilation.

Ventilation Efficacy. Numerous studies have evaluated the ability of rescuers to provide ventilation to models using BVM. Results have uniformly shown that one-rescuer BVM is ineffective.^{17,44-46,60,70,71}

These reports suggest diminished volume delivery due to the inability of a single rescuer to use one hand to hold the mask securely while maintaining airway patency

(head-tilt, chin-lift) and squeezing the bag with the other hand. Results from these studies vary widely, with a minimum value reported by Hess et al⁴² of 300 mL up to 700 mL reported by Seidelin et al.⁴⁵ Comparison of the results is likewise a problem. Models used for BVM research include resuscitation and intubation mannequins connected to test lungs that vary from the very simple to complex, precision test lungs. A Laerdal Resusci-Anne resuscitation mannequin requires alignment of airway structures prior to providing ventilation, whereas the intubation head has a preformed open airway that eliminates the need for airway opening maneuvers. It is obvious that results using these two models might be quite different. Test lungs can cause similar problems. With a training test lung, resistance and compliance can be set, whereas the lungs inside a resuscitation mannequin are essentially two bags. Additional variability may be due to differences in volume-monitoring sophistication; some studies use pneumotachographs and others respirometers. However, even with the wide variability in results, it can easily be concluded that BVM by one rescuer is typically ineffective.

Several suggestions have been made to improve ventilation provided by BVM. Cummins et al⁷² and Barnes and Adams⁷³ have evaluated the face-and-thigh-squeeze technique (FATS). This method is used in prehospital care to increase delivered volume. The rescuer kneels and places the victim's head between his knees. One hand holds the mask and provides the head-tilt and jaw-lift by pulling up on the mandible. The remaining hand squeezes the bag against the rescuer's thigh in an attempt to maximize bag deflation and, thus, volume delivery.

Volume delivery during BVM can also be increased by using two rescuers.⁷⁴ Similarly, when two hands are used to squeeze the bag, tidal volume is enhanced. Hess and Baran⁴² found delivered volume almost doubled when using two-person BVM and two-hand compression of the bag (from 300 mL to 580 mL). However, in the prehospital setting the availability of two rescuers to perform ventilation is unlikely.

Other factors affecting BVM performance include mask design,⁷⁵ respiratory impedance,⁷⁶ hand size,^{77,78} and volumetric feedback.⁷⁹ In general, a soft, pliable, inflatable, sealing mask improves the face-to-mask seal and increases V_T . As pulmonary compliance falls or resistance increases, delivered volume falls, and larger hands are associated with larger V_T s. Volumetric feedback improves volume delivery by allowing the rescuer to get a feel for adequate volume delivery.

We have recently recorded breath-by-breath volumes during bag-valve-endotracheal tube ventilation during CPR in the emergency department.⁸⁰ Our results show average V_T s during CPR of 571 mL at a frequency of 24 breaths/min, with average peak airway pressures of 41 cm H_2O , and aver-

age inspiratory times of 1.1 seconds. These values are considerably different from AHA guidelines and suggest that some type of volume and/or frequency monitor might improve the efficiency of bag-valve ventilation.

Oxygen Concentration. Numerous reports have evaluated the ability of resuscitators to deliver a desired oxygen concentration.⁸¹⁻⁹⁰ According to ASTM⁸ and ISO,⁹ F_{DO_2} should be ≥ 0.85 with an oxygen reservoir in use and an oxygen flow of 15 L/min. The AHA recommends an F_{DO_2} of 1.0 or "the highest possible oxygen concentration should be administered as soon as possible to all patients with cardiac or respiratory arrest."¹ This recommendation seems to be prudent, but no studies have proven an F_{DO_2} of 1.0 to be superior to an F_{DO_2} of 0.50. In fact, it could be speculated that an F_{DO_2} of 1.0 might accentuate reperfusion injury. The required, safe F_{IO_2} during CPR is a matter of some controversy and requires further sound scientific investigation for clarification.

Instances in which F_{DO_2} s of ≤ 0.85 have been reported with bag-valve devices are related to high minute ventilation, insufficient reservoir volume, and ambient temperatures $\leq 0^\circ\text{C}$.⁸¹⁻⁹⁰ Most current resuscitator designs are capable of delivering a minimum F_{DO_2} of 0.85 during CPR.

Nonbreathing Valve. The NRV is the heart of the resuscitation bag. According to ASTM, the NRV should resist breakage, function in the presence of vomitus, allow flows of up to 30 L/min without sticking, and continue to function at extremes of temperature (-18°C to $+50^\circ\text{C}$).⁸

Studies of NRV competence in the presence of vomitus have shown adequate performance in a majority of cases.⁸¹⁻⁹⁰ Several devices have failed at low temperatures when condensation froze the valve's components.⁹⁰ Reports have also shown that many devices fail the "drop test" of falling one meter to a concrete floor.⁸⁷⁻⁹⁰

Back-leak through an NRV should be minimal to prevent rebreathing, and most devices have few problems with valve incompetence. Several cases of NRV failure due to misassembly have been reported, which underscores the need for operator vigilance and a design that eliminates the possibility of misassembly.⁹¹

Hess and Simmons⁹² found that resistance to flow of NRV in adult resuscitators is variable. They found three devices that exceeded the 5-cm- H_2O back-pressure standard at a flow of 50 L/min, specified by ASTM⁸ (Fig. 11). Hess and Simmons⁹² suggested that the increased flow resistance of some NRVs could prolong expiration and cause air trapping, which might lead to barotrauma or further impediment to ventilation. They also suggested that NRVs with high flow resistance might contribute to fatigue of the rescuer squeezing the bag.

Spontaneous breathing through the NRV of resuscita-

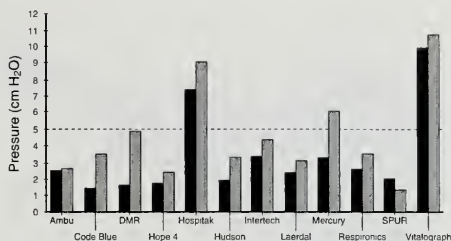


Fig. 11. Measured inspiratory (■) and expiratory (▒) back pressure across 12 valves at flow of 50 L/min, at which back pressure should be < 5 cm H_2O .

tors has been an area of some controversy.^{93,94} In 1994, Hess and colleagues evaluated the imposed work of breathing created during simulated spontaneous breathing via the NRV of 11 disposable resuscitators.⁹⁵ They found that both inspiratory and expiratory work of breathing is elevated. Imposed work of breathing increases with the application of PEEP and with increased patient flow demand. Measurements of work in this study were quite high (up to 2.0 J/L) and represent a 10- to 100-fold increase compared to the imposed work of common intensive care ventilators.⁹⁵ The authors recommended that the practice of allowing spontaneous breathing via disposable resuscitation bags be abandoned.

BVM ventilation remains the most popular method of ventilatory support by BLS providers in the field.⁹⁶ Many factors previously discussed allow BVM ventilation to be used extensively despite evidence of its limitations. More effective provider training may be the answer to problems with BVM, rather than introduction of new ventilatory techniques.

Oxygen-Powered Breathing Devices

Description

Oxygen-powered breathing devices (OPDs) are frequently called demand valves or known by a trade name such as Flynn or Elder valve. Both ASTM and ISO have created standards for OPD.^{8,9} A typical OPD consists of a demand valve that can be manually or patient triggered (Fig. 12). The OPD is connected to a 50 psig source of gas and is attached to the patient via a standard 15-22 mm connector. During manual activation of the demand valve, the operator depresses the actuator, allowing gas to travel to the patient. According to the ASTM standard, this flow should be restricted to 40 L/min to prevent excessive ventilation and gastric distension. Modern OPDs also limit the available airway pressure (via a pressure-limiting valve) and the length of inspiratory time. Inspiration is terminated

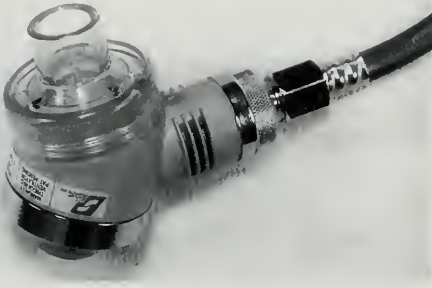


Fig. 12. (A) manually triggered oxygen-powered breathing device.

after 3 seconds, even if the operator continues to depress the actuator. During spontaneous breathing, the demand valve is pressure triggered and allows a flow of up to 100 L/min on patient demand.

Assessment & Limitations

The history of the OPD is troubling. A number of studies have shown failure of the pressure-limiting devices within the OPD and subsequent high airway pressures (> 100 cm H_2O).⁹⁷⁻⁹⁹ Pradis and Caldwell have reported a case of pneumocephalus resulting from OPD-to-mask ventilation, caused by excessive airway pressures. However, some of this work^{97,99} was done prior to introduction of the 40L/min flow-limiting design.

More recently, Menegazzi and colleagues^{100,101} have evaluated the use of the OPD in a model, providing ventilation via OPD to mask and OPD to endotracheal tube. Using a mannequin system similar to that developed by our group, Menegazzi et al found no difference in delivered V_T s between BVM and OPD-to-mask ventilation. They did however, report significantly increased gastric insufflation with BVM ventilation. This difference was attributed to the high inflation pressures and flows that can be developed during BVM ventilation compared to the fixed steady flow of 40 L/min provided by the OPD. Menegazzi and Winslow¹⁰⁰ suggested that OPD-to-mask was superior to BVM ventilation in preventing gastric insufflation in this model; however, they suggested that clinical studies be performed prior to widespread use of the OPD.

Mesezzo et al¹⁰¹ compared the OPD to a bag-valve-device for ventilation of a model via an endotracheal tube. They found similar delivered V_T s at all levels of compliance tested. Additionally, they reported a higher airway pressure with the bag-valve device compared to the OPD (31 cm H_2O vs 13 cm H_2O at normal lung compliance). The authors¹⁰¹ suggested that the lower airway pressures

were due to the fixed 40 L/min inspiratory flow and that demand valve ventilation through an endotracheal tube may be preferable. One limitation of the OPD is the inability of the rescuer to gauge patient compliance.

The history of the OPD is a major limitation to adopting its use today. However, devices that meet the ASTM standard can probably be used safely and effectively. Clinical studies of the OPD in the field are necessary prior to recommending widespread use. AHA rates the OPD as Class II.

Ventilators for Resuscitation

Description

Transport ventilators for prehospital care and CPR are typically time-triggered, volume-limited, time-cycled devices.^{102,103} They are typically simple, having one or two controls, providing only one mode, no PEEP, and a F_{DO_2} of 1.0. The AHA has suggested that automatic transport ventilators meeting the following criteria are Class I devices:¹

- Have a lightweight connector with a standard 15/22 mm fitting
- Have a lightweight (2-5 kg), compact, rugged design
- Operate in all environmental conditions and extremes of temperature
- Have a peak pressure limit of 60 cm H_2O , with an option of 80 cm H_2O
- Have an audible alarm when the peak-pressure limit is exceeded
- Have minimal gas consumption
- Have minimal gas-compression volume in the circuit
- Deliver an fractional oxygen concentration of 1.0
- Have an inspiratory time of 2.0 seconds for adults and 1.0 second for children with corresponding peak inspiratory flowrates of 30 and 15 L/min.
- Have at least two breathing frequency settings.

Additionally, AHA lists desirable features that include a manometer, separate rate and volume controls, and a low pressure alarm.

Characteristics

Our group¹⁰³ has published descriptions of prehospital ventilators and desirable characteristics. These are similar to those listed by AHA.

Operational Characteristics. All ventilators used in prehospital care should be time or volume cycled. Modified pressure-cycled devices have been used by skilled clinicians, although their general use is discouraged. Constant flowrate ventilators are preferred, but nonconstant flow ventilators can be used successfully. Inspiratory flowrate and V_T should be minimally affected by changes in air-

ways resistance and lung-thorax compliance ($< 10\%$ change). The ideal is that gas consumption to power the ventilator's system components be zero; however, low-level gas consumption ($< 5 \text{ L/min}$) is acceptable, but decreases the ventilator's operating time.

Portability. Size and weight are the chief concerns. Orientation of controls and handles for carrying the device are also important. A weight less than 4 kg is considered desirable.

Power Source. Pneumatically powered ventilators are preferable to electronically powered ones. Ventilators that require pneumatic and electronic power sources require compressed oxygen and a direct current power source (battery); thus, two perishable power sources are necessary. There are tradeoffs, however, with these two types of ventilators. Electronically powered ventilators provide more precise control of variables and do not consume gas during operation. Either type of ventilator, thus, can be adequate.

Ease of Operation. Operation of the ventilator should be as simple as possible, with a minimum number of controls. Each control should be labeled as to function, effects, and possible hazards. When possible, a diagram of the circuit and its proper connection to the patient should be printed on the side or back panel of the ventilator. Ventilators used in prehospital care do not need a variable F_{DO_2} , continuous positive airway pressure (CPAP), or a variety of ventilatory modes. In our study of prehospital ventilatory support, 95% of patients remained apneic during transport. Addition of a demand-flow valve adds size and weight to the ventilator and can also increase gas consumption. Some transport ventilators position the demand-flow valve on the endotracheal tube. This can result in inadvertent extubation or kinking of the tube due to the weight of the demand-flow valve. As stated previously, independent control knobs for V_T and f should be available. An important, and often overlooked, feature is the manual inhalation control. This allows the operator to control ventilator rate and V_T manually, independent of the ventilator's settings, in special situations. This control is also helpful during auscultation of breath sounds to confirm proper endotracheal tube placement.

Assembly and Disassembly. The ventilator breathing circuit and exhalation valve should be simple, and incorrect assembly should be impossible. Likewise, high-pressure tubing from the gas source to the ventilator and from ventilator to patient should not be interchangeable—to avoid misassembly. If a valve is used to control the flow of inspiratory and expiratory gases (such as an NRV), it should be easily cleaned of vomitus, blood, and secretions.

Safety. The number of safety devices are limited by the small size of transport ventilators. A high-pressure relief valve that vents gas to the atmosphere at a preselected peak inflation pressure, or PIP (typically 60-80 cm H_2O for adults and 30-40 cm H_2O for children), should be required on transport ventilators. Activation of the high pressure limit should be signaled by a visual or audible alarm to alert the operator. The ventilator should also have an anti-asphyxia valve. If gas and/or electrical power supplies are exhausted, the system should allow the patient to breathe ambient air without excessive resistance. Battery-powered ventilators should be equipped with a low-battery signal that indicates when 1 hour of power remains. Loss of oxygen power supply to a transport ventilator should activate audible and/or visual alarms.

Durability. Extremes of temperature and humidity should not adversely affect the operation of transport ventilators. These ventilators should be capable of operating on a moment's notice (even after prolonged periods of storage) and should be able to withstand rough treatment and still operate properly. Occasionally, transport ventilators are used in hazardous environments and should, therefore, have protective cases that can withstand erosion by chemicals or other foreign substances.

Maintenance. Preventive maintenance should be performed periodically by the manufacturer or a trained technician. Routine maintenance should be minimal.

Breathing Valve. One distinct feature incorporated into ventilators used in prehospital care is a breathing or inhalation/exhalation-valve mechanism. This valve is connected to the ventilator by corrugated or high pressure tubing and directs inspiratory and expiratory gases to and from the patient. The valve may be simple, such as an NRV, or it can be complicated. Some of these valves may house an NRV, a pressure-limiting valve, a demand-flow valve, and an anti-asphyxia valve. As a result, several connecting ports on these breathing valves are required to accommodate additional valves. The connecting ports should have different inside and outside diameters such that improper connection cannot occur. Resistance to inhalation and exhalation should be low $< 3 \text{ cm H}_2\text{O} \cdot \text{s} \cdot \text{L}^{-1}$.

The weight of the breathing valve should be minimal ($< 0.3 \text{ kg}$), because heavy or awkward valves can cause kinking of the endotracheal tube or accidental extubation. Cleaning the valve should be an easy and simple operation.

Assessments & Comparisons

Evaluations of the ability of transport ventilators to deliver adequate V_T s have been published.¹⁰⁴⁻¹⁰⁷ These stud-

ies, however, typically deal with a model of an intubated patient. We have presented data regarding use of transport ventilators in the prehospital environment but also only in intubated patients.⁹⁶

We found that a transport ventilator with an adjustable flow control delivered more consistent V_T s than does BVM ventilation. The ventilator provided smaller V_T s than mouth-to-mouth or mouth-to-mask ventilation but produced markedly less gastric insufflation than either of those techniques.⁴² A ventilator with a fixed flow provided smaller V_T s.

Greenslade¹⁰⁸ compared volumes delivered to a mannequin with mouth-to-mouth, mouth-to-mask, BVM, and ventilator-to-mask ventilation during one-rescuer CPR. This study had rescuers perform CPR with each ventilation technique in the back of a moving ambulance. Data from this study are shown in Tables 3 and 4. Greenslade found that mouth-to-mouth ventilation allowed the largest minute ventilation and most successful number of compressions per minute. Use of the ventilator and manual resuscitator resulted in the smallest minute ventilation and lowest number of compressions. He recommended that mouth-to-mask ventilation be used in unintubated patients and that ventilators be reserved for intubated patients.

Table 3. Summary Statistics for Minute Volume (L) for 17 Subjects*

Method/Device	Volume Mean (SD)	Minimum Volume	Maximum Volume
Mouth-to-mouth	10.5 (3.1)	4.6	17.9
Mouth-to-mask	8.5 (2.7)	4.6	15.6
Ambu resuscitator	4.5 (1.2)	2.4	6.2
PneumPAC resuscitator	4.7 (1.0)	3.2	6.4

*From Reference 108, with permission.

Table 4. Summary Statistics for Compressions per Minute for 17 Subjects*

Ventilation Method	Number/min (SD)	Minimum Number	Maximum Number
Mouth-to-mouth	40.3 (7.8)	29	57
Mouth-to-mask	38.2 (7.1)	26	54
Ambu resuscitator	36.2 (5.5)	28	47
PneumPAC resuscitator	33.1 (5.4)	25	44

*From Reference 108, with permission.

Greenslade further suggested "keeping ambulance speed below 30 mph and using oxygen-supplemented mouth-to-mask ventilation, appear to offer the best chance of success in unintubated patients receiving single-operator CPR."¹⁰⁸

We agree with Greenslade and would recommend BLS

squads perform oxygen-supplemented mouth-to-mask ventilation until endotracheal intubation is performed, after which time bag-valve, OPD, or ventilator work effectively. One advantage of the ventilator is that it frees a rescuer to perform other tasks. A disadvantage may be the loss of 'feel' for pulmonary compliance.

We are also puzzled by the AHA recommendation for a ventilator with a fixed flow of 30 L/min. Because the ventilator is most often used in intubated patients, there is no advantage to a slow inspiratory flow. In fact, the slow inspiratory flow creates two distinct disadvantages:

1. It makes it impossible to make up for leaks around a mask if used in an unintubated patient.

2. If a higher frequency is desired (ie, for head injury), air trapping may result due to insufficient expiratory time.

It is our contention that while a fixed flow setting may be useful, the ventilator should have a provision for increasing flow.

FUTURE RESEARCH

We believe future research into emergency ventilation should consider the level of ventilation provided and reassess the current frequency and tidal volume standards. Because the current standards are based on Safar's study of paralyzed, live volunteers,⁵⁷ it might be reasonable to assume a patient suffering cardiac arrest would have a lower CO_2 production and require less minute ventilation. A reassessment of the necessary F_{DO_2} is also in order.

With new CPR techniques, (eg, vest CPR, active compression-decompression) ventilatory techniques may require further modification and as is suggested elsewhere in the proceedings of this conference,¹⁰⁹ perhaps ventilation isn't necessary at all.

REFERENCES

1. Emergency Cardiac Care Committee and Subcommittees, American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiac care. *JAMA* 1992;268:2171-2302.
2. Emergency Care Research Institute. Manually operated resuscitators. *Health Devices* 1971;1:13-17.
3. Emergency Care Research Institute. Manual resuscitators. *Health Devices* 1979;8:133-146.
4. Emergency Care Research Institute. Gas-powered resuscitators. *Health Devices* 1978;8:24-38.
5. Emergency Care Research Institute. Gas-powered pulmonary resuscitators. *Health Devices* 1989;18:362-363.
6. Emergency Care Research Institute. Pulmonary resuscitators. *Health Devices* 1988;17:348-354.
7. Emergency Care Research Institute. Exhaled air pulmonary resuscitators (EAPR's) and disposable manual pulmonary resuscitators (DMPR's). *Health Devices* 1989;18:333-352.
8. American Society of Testing Materials. Standard specifications for minimum performance and safety requirements for resuscita-

- tors intended for use with humans. Designation F 920-85. Philadelphia, ASTM, 1985.
9. International Standards Organization. International Standard ISO 8382:1988(E): Resuscitators intended for use with humans. New York, American National Standards Institute, 1988.
10. Ruben H, Knudsen EJ, Carugli G. Gastric insufflation in relation to airway pressure. *Acta Anaesthesiol Scand* 1961;5:107-114.
11. Melker RJ. Recommendations for ventilation during cardiopulmonary resuscitation: Time for a change? *Crit Care Med* 1985;13:882-883.
12. Fillmore SJ, Shapiro M, Killip J. Serial blood gas studies during cardiopulmonary resuscitation. *Ann Intern Med* 1970;72:465.
13. Gilston A. Clinical and biochemical aspects of cardiac resuscitation. *Lancet* 1965;2:1039-1043.
14. Himmelhoch SR, Dekker A, Gazzaniga AB, Like AA. Closed-chest cardiac resuscitation: A prospective clinical and pathological study. *N Engl J Med* 1964;270:118-122.
15. Ornato JP, Bryson BL, Donovan PJ, Farquharson RR, Jaeger C. Measurement of ventilation during cardiopulmonary resuscitation. *Crit Care Med* 1983;11:79-82.
16. Davis K, Johnson RC, Branson RD, Campbell RS, Johnson DJ, Tabor T. Pulmonary compliance of cardiac arrest victims. A reassessment (abstract). *Crit Care Med* 1994;22:S135.
17. Melker RJ, Banner MJ. Ventilation during CPR: Two-rescuer standards reappraised. *Ann Emerg Med* 1985;14:397-402.
18. Sellick BA. Cricoid pressure to control regurgitation of stomach contents during induction of anesthesia. *Lancet* 1961;2:404-406.
19. Moynihan RJ, Brock-Utne JG, Archer JH, Feld LH, Kreitzman TR. The effect of cricoid pressure on preventing gastric insufflation in infants and children. *Anesthesiology* 1993;78:652-656.
20. Vanner RG, O'Dwyer JP, Pryle BJ, Reynolds F. Upper oesophageal sphincter pressure and the effect of cricoid pressure. *Anesthesia* 1992;47:95-100.
21. Ralph SJ, Wareham CA. Rupture of the oesophagus during cricoid pressure. *Anaesthesia* 1991;46:40-41.
22. Barker SJ, Karagianes T. Gastric barotrauma: A case report and theoretical considerations. *Anesth Analg* 1985;64:1026-1028.
23. II Kings 4:31-35 (KJV).
24. Hess D, Kapp A, Kurtke W. The effect of delivered oxygen concentration of the rescuer's breathing supplemental oxygen during exhaled gas ventilation. *Respir Care* 1985;30:691-694.
25. Rottenberg EM, Dzwonczyk R, Reilly TE, Malone M. Use of supplemental oxygen during bystander-initiated CPR. *Ann Emerg Med* 1994;23:1027-1030.
26. Ornato JP, Hallagan LF, McMahon SB, Rostafinski A. Attitudes of BCLS instructors about mouth-to-mouth resuscitation during the AIDS epidemic. *Ann Emerg Med* 1990;19:151-156.
27. Pane GA, Salsness KA. A survey of participants in a mass CPR training course. *Ann Emerg Med* 1987;16:1112-1116.
28. Link RN, Feingold AR, Charap MH, Freeman K, Shelou SP. Concerns of medical and pediatric house officers about acquiring AIDS from their patients. *Am J Public Health* 1988;78:455-459.
29. American Heart Association. Supplemental guidelines: Risk of infection during CPR training and rescue. *JAMA* 1989;262:22714-2715.
30. Fluck RR, Sorbello JG. Mouth-to-mouth resuscitation by lay rescuers - should they or shouldn't they? *Respir Care* 1990;35:831-832.
31. Durbin CG. Mouth-to-mask resuscitation by lay rescuers: - will they or won't they? *Respir Care* 1990;35:832-834.
32. Heilman KM, Muscheheim C. Primary cutaneous tuberculosis resulting from mouth-to-mouth respiration. *N Engl J Med* 1965;273:1035-1036.
33. Feldman HA. Some recollections of the meningococcal disease. *JAMA* 1972;220:1107-1112.
34. Hendricks AA, Shapiro EP. Primary herpes simplex infection following mouth-to-mouth resuscitation. *JAMA* 1980;243:257-258.
35. Finkelhor RS, Lampman JH. Herpes simplex infection following cardiopulmonary resuscitation. *JAMA* 1980;243:650.
36. Harris MJ, Wendel RT. Transmission of herpes simplex during cardiopulmonary resuscitation. *Compreh Ther* 1984;10:15-17.
37. Todd MA, Bell JS. Shigellosis from cardiopulmonary resuscitation. *JAMA* 1980;243:331.
38. Ahmad F, et al. Transmission of salmonella via mouth-to-mouth resuscitation. *Lancet* 1990;335:787.
39. Mamby SA. A plea for CPR equipment in public places. *N Engl J Med* 1990;322:1161.
40. Hodgins L. New law in the Big Apple mandates CPR infection control equipment. *Occupational Health and Safety* 1992;61:56-58.
41. Sainsbury DA, Davis R, Walker MC. Artificial ventilation for cardiopulmonary resuscitation. *Med J Aust* 1984;141:509-511.
42. Hess D, Baran C. Ventilatory volumes using mouth-to-mouth, mouth-to-mask, and bag-valve-mask techniques. *Am J Emerg Med* 1985;3:292-296.
43. Lawrence PJ, Sivaneswaran N. Ventilation during cardiopulmonary resuscitation: which method? *Med J Aust* 1985;143:443-446.
44. Harrison RR, Maul KI, Keenan RL, Boyan CP. Mouth-to-mask ventilation: A superior method of rescue breathing. *Ann Emerg Med* 1982;11:74-76.
45. Seidelin PH, Stolarek JH, Littlewood DG. Comparison of six methods of emergency ventilation. *Lancet* 1986;2:1274-1275.
46. Johannigman JA, Branson RD, Davis K, Hurst JM. Techniques of emergency ventilation: A model to evaluate Vt, airway pressure, and gastric insufflation. *J Trauma* 1991;31:93-98.
47. Waters RM. Simple methods for performing artificial respiration. *JAMA* 1943;123:559-561.
48. Don Michael A, Forrester JS. Mouth-to-mouth ventilation: The dying art. *Am J Emerg Med* 1992;10:156-161.
49. Lightsey DM, Shah PK, Forrester JS, Don Michael A. A human immunodeficiency virus resistant airway for cardiopulmonary resuscitation. *Ann J Emerg Med* 1992;10:73-77.
50. Rossi R, Ahnefeld FW. Expired air resuscitation in emergency situations with Ambu-Life key. *Notfallmedizin* 1990;16:3-7.
51. Baskett PJF. Advances in cardiopulmonary resuscitation. *Br J Anaesth* 1992;69:182-193.
52. Westfall MD. Kiss of Life mask: Evaluation or opinion? (Letter) *Am J Emerg Med* 1992;10:616.
53. Rossi R, Lindner KH, Ahnefeld FW. Devices for expired air resuscitation. *Prehospital Disaster Med* 1993;8:123-126.
54. Simmons M. Resistance to flow through the valves of three face shield CPR barrier devices (abstract). *Respir Care* 1994;39:1068.
55. Simmons M, Deao D, Moon L. An evaluation of three face shields for volume delivery during rescue breathing (abstract). *Respir Care* 1993;39:1068.
56. Elam JO, Brown ES, Elder JD Jr. Artificial respiration by mouth-to-mask method. *N Engl J Med* 1954;250:749-754.
57. Safar P, McMahon M. Mouth to airway emergency artificial respiration. *JAMA* 1958;166:1459-1461.
58. Safar P. Pocket mask for emergency artificial ventilation and oxygen inhalation. *Crit Care Med* 1974;2:273-276.
59. Thomas AN, Weber EC. A new method of two-resuscitator CPR. *Resuscitation* 1993;26:173-176.
60. Thomas AN, O'Sullivan K, Hyatt J, Barker SJ. A comparison of bag mask and mouth mask ventilation in anaesthetized patients. *Resuscitation* 1993;26:13-21.
61. Tolley PM, Watts J, Hickman JA. Comparison of the use of the laryngeal mask and face mask by inexperienced personnel. *Brit J Anaesth* 1992;69:320-321.

62. Thomas AN, Bergesio R, Hyatt J, Barker SJ. Mouth mask ventilation: Use of a Pall ulipor breathing system and effect of mask design (abstract). *Brit J Anaesth* 1992;69:527P-528P.
63. Johannigman JA, Branson RD. Oxygen enrichment of expired gas for Mouth-to-mask resuscitation. *Respir Care* 1991;36:99-103.
64. Stahl JM, Cutfield GR, Harrison GA. Alveolar oxygenation and mouth-to-mask ventilation: effects of oxygen insufflation. *Anaesth Intensive Care* 1992;20:177-186.
65. Thomas AN, Hyatt J, Chen JL, Barker SJ, The Laerdal pocket mask: effects of increasing supplementary oxygen flow. *Anaesthesia* 1992;47:967-971.
66. Palmisano JM, Moler FW, Galura C, Gordon M, Custer JR. Influence of Vt, respiratory rate, and supplemental oxygen flow on delivered oxygen fraction using a mouth-to-mask ventilation device. *J Emerg Med* 1993;11:685-689.
67. Hess D, Ness C, Oppel A, Rhoads K. Evaluation of Mouth-to-mask ventilation devices. *Respir Care* 1989;34:191-195.
68. Hess DR. Manual and gas powered resuscitators. in Branson RD, Hess DR, Chatburn RL (eds) *Respiratory Care Equipment*. J.B. Lippincott, 1995 (in press).
69. Barnes TA. Emergency ventilation techniques and related equipment. *Respir Care* 1992;37:673-694.
70. Elling R, Politis J. An evaluation of emergency medical technicians ability to use manual ventilation devices. *Ann Emerg Med* 1983;12:765-768.
71. Giffen PR, Hope CE. Preliminary evaluation of a prototype tube-valve-mask ventilator for emergency artificial ventilation. *Ann Emerg Med* 1991;20:262-266.
72. Cummins RO, Austin D, Graves JR, Litwin PE, Pierce J. Ventilation skills of emergency medical technicians: A teaching challenge for emergency medicine. *Ann Emerg Med* 1986;15:1187-1192.
73. Barnes TA, Adams G. Ventilatory volumes using mouth-to-mouth, bag-valve-mask, and pocket face mask (abstract). *Respir Care* 1991;36:1292.
74. Jesudian MC, Harrison RR, Keenan RL, Maul K. Bag-valve-mask ventilation: two rescuers are better than one; preliminary report. *Crit Care Med* 1985;13:122-123.
75. Stewart RD, Kaplan R, Penrock B, Thompson F. Influence of mask design on bag-mask ventilation. *Ann Emerg Med* 1985;14:403-406.
76. Hess D, Goff G. The effects of two-hand versus one-hand ventilation on volumes delivered during bag-valve ventilation at various resistances and compliances. *Respir Care* 1987;32:1025-1028.
77. Augustine JA, Seidel DR, McCabe JB. Ventilation performance using a self-inflating anesthesia bag: effect of operator characteristics. *Am J Emerg Med* 1987;5:267-270.
78. Hess D, Goff G, Johnson K. The effect of hand size, resuscitator brand, and use of two hands on volumes delivered during adult bag-valve ventilation. *Respir Care* 1989;34:805-810.
79. Powers WE. Evaluation of a training method that uses volumetric feedback with bag-valve-mask ventilation techniques (abstract). *Respir Care* 1988;33:942-943.
80. Branson RD, Davis K, Johnson RC, Campbell RS, Tabor T. Manual ventilation during cardiopulmonary resuscitation (abstract). *Respir Care* 1994;39:1068.
81. Priano L, Ham J. A simple method to increase FDO₂ of resuscitator bags. *Crit Care Med* 1978;6:48-49.
82. Fitzmaurice MW, Barnes TA. Oxygen delivery performance of three adult resuscitation bags. *Respir Care* 1980;25:928-933.
83. LeBovet L. 1980 assessment of eight adult manual resuscitators. *Respir Care* 1980;25:1136-1142.
84. Barnes TA, Watson MW. Oxygen delivery performance of four adult resuscitation bags. *Respir Care* 1982;27:139-146.
85. Barnes T, Watson M. Oxygen delivery performance of old and new designs of the Laerdal, Vitalograph, and AMBU adult manual resuscitators. *Respir Care* 1983;28:1121-1128.
86. Phillips GD, Showronski GA. Manual resuscitators and portable ventilators. *Anaesth Intensive Care* 1986;14:306-313.
87. Campbell TP, Stewart RJ, Kaplan RM, DeMichiel RV, Morton R. Oxygen enrichment of bag-valve-mask units during positive-pressure ventilation: a comparison of various techniques. *Ann Emerg Med* 1988;17:232-235.
88. Barnes TA, Potash R. Evaluation of five disposable operator powered adult resuscitators. *Respir Care* 1989;34:254-261.
89. Barnes TA, McGarry W. Evaluation of ten disposable manual resuscitators. *Respir Care* 1990;35:960-968.
90. Barnes TA, Stockwell DL. Evaluation of ten manual resuscitators across an operational temperature range of -18°C to 50°C. *Respir Care* 1991;36:161-172.
91. Munford BJ, Wishaw KJ. Critical incidents with non-rebreathing valves. *Anaesth Intensive Care* 1990;18:560-563.
92. Hess D, Simmons M. An evaluation of the resistance to flow through the patient valves of twelve adult manual resuscitators. *Respir Care* 1992;37:432-438.
93. Mills PJ, Baptiste J, Preston J, Barnes GM. Manual resuscitators and spontaneous ventilation: An evaluation. *Crit Care Med* 1991;19:1425-1431.
94. Stemp LJ. Manual resuscitators and spontaneous ventilation - an evaluation (letter). *Crit Care Med* 1992;20:1496.
95. Hess D, Hirsch C, Marquis-D'Amico C, Kacmarek R. Imposed work and oxygen delivery during spontaneous breathing with adult disposable manual ventilators. *Anaesthesiology* 1994;81:1256-1263.
96. Johannigman JA, Branson RD, Johnson DJ, Hurst JM. Techniques of airway management and ventilatory support in pre-hospital care: Patterns of practice and the use of transport ventilators. *Academic Emergency Medicine* 1995 (in press).
97. Osborn HH, Kaye D, Horne H, Bray W. Excess ventilation with oxygen powered resuscitators. *Am J Emerg Med* 1984;2:408-413.
98. Pradis IL, Caldwell EJ. Traumatic pneumocephalus: a hazard of resuscitators. *J Trauma* 1979;19:61-63.
99. Fasi TH, Lucas BG. An evaluation of some mechanical resuscitators for use in the ambulance service. *Ann R Coll Surg Engl* 1980;62:291-293.
100. Menegazzi JJ, Winslow HJ. In-vitro comparison of bag-valve-mask and the manually triggered oxygen powered breathing device. *Acad Emerg Med* 1994;1:29-33.
101. Mesezzo VN, Lukitsch K, Menegazzi J, Mosesso J. Comparison of delivered volumes and airway pressures when ventilating through an endotracheal tube with bag valve versus demand valve. *Prehosp Disaster Med* 1994;9:24-28.
102. Branson RD. Intrahospital transport of critically ill mechanically ventilated patients. *Respir Care* 1992;37:775-795.
103. Branson RD, McGough EK. Transport ventilators. In: Banner MJ (ed), *Problems in critical care: positive pressure ventilation*. Philadelphia PA: JB Lippincott 1990:254-274.
104. Nolan JP, Baskett JF. Gas-powered and portable ventilators: an evaluation of six models. *Prehosp Disaster Med* 1972;7:25-34.
105. McGough EK, Banner MJ, Melker RJ. Variations in V_T with portable transport ventilators. *Respir Care* 1992;37:233-239.
106. Campbell RS, Davis K, Johnson DJ, Porembska D. Laboratory and clinical evaluation of the Impact 750 portable ventilator. *Respir Care* 1992;37:29-36.
107. Johannigman JA, Branson RD, Campbell RS, Hurst JM. Laboratory and clinical evaluation of the MAX transport ventilator. *Respir Care* 1990;35:952-959.
108. Greenslade GL. Single operator cardiopulmonary resuscitation in

Branson Discussion

Barnes: Rich, that was a great review. This question about the size of tidal volume and the minute volume during CPR is an interesting question. I recently came across a paper published in 92 by Popkin and Swenson¹ titled "Resuscitation from Severe Acute Hypercapnia" where the patient was well-oxygenated, but the P_{aCO_2} rose to 373 torr, probably not the true value that high in the scale—but the point was this patient was ventilated after a dental procedure during which this extreme level of hypercarbia developed and made a complete recovery, even though his pH was down to 6.6 and his P_{aCO_2} was really extremely high. It raises the whole question of how necessary is ventilation during CPR when there's no blood flow, if the patient is well oxygenated. It may be that a small tidal volume and small minute ventilation with good oxygenation is all that is needed.

1. Popkin RT, Swenson ER. Resuscitation from severe acute hypercapnia. Determinants of tolerance and survival. Chest 1992;102(6):1742-1745.

Branson: I can't agree more Tom. We are just now embarking on a study to measure CO_2 production in patients undergoing CPR, to try to determine what minute ventilation is required to maintain a normal alveolar CO_2 . I'm sure all of us around the table who do ARDS research know that the new trend is permissive hypercapnia. We let the patient's P_{aCO_2} be 80, 90, or 100 and the pH 7.25 or 7.20, and we don't worry about it. It may be that this whole CPR issue of mechanical ventilation has been overstated. We do have to be careful because after near-drowning and in pediatric patients, ventilation and oxygenation, as we were told this morning, are the Number 1 thing to do. But in adult CPR, ventilation may

not be necessary at all. It may just be that we have to provide a continuous tracheal insufflation of oxygen. With things like active decompression of the chest, we may just have to intubate the patient, run a flow of oxygen down the trachea, and not have to ventilate the patient at all.

Bishop: I'd just like to go along with the same theme—that, in fact, ventilation may not only not be necessary but in certain patients it can be a hazard. Those are patients with obstructive airways disease. I've seen deaths in patients who were just barely compensating, who, once they were intubated and positive-pressure ventilation started, had no blood pressure obtainable, basically because of air trapping and a lack of venous return. I've been an expert witness in a case in which the patient was intubated but never again had a pulse. EMD occurred because of air trapping. Yet, unless you stand there and say "Take it easy. Don't ventilate like that," everybody's first reaction, as soon as that tube is in, is to go like crazy with the bag.

Branson: We have published an abstract for the AARC OPEN FORUM that reports in the course of a compliance study, the breath-to-breath measurements over the entire period of CPR in the emergency department—an average respiratory rate during resuscitation of about 21 breaths per minute. The average tidal volume was somewhere around 600 mL.¹ Dean and I have both worked with a couple of manufacturers, trying to convince them of the importance of some type of feedback to the rescuer—to tell them what they're doing, even if it's just a light that turns green when they are doing it right and turns red when they are doing it wrong. I think you're right. Caregivers overventilate the patient in terms of rate and underventilate the patient with respect to tidal volume.

1. Branson R, Davis K Jr, Johnson R, Campbell R, Tabor T. Manual ventilation during cardiopulmonary resuscitation. Respir Care 1994;39(11):1068.

Bishop: Intubation is often a preterminal event in severe obstructive airways disease, and I think it's because of positive-pressure ventilation and air trapping.

Hamill: I have two comments. One, we can take patients coming into the operating room—the classic situation is during the placement of double-lumen tubes—we're so busy trying to check the position of the tube and squeezing the bag that most of those people end up with systolic pressures in the range of 50 to 70 in spite of fluid loading prior to the event. Take a patient who is hypovolemic, if we get a little enthusiastic, it's clearly going to make a big difference. The other comment I wanted to make was about cricoid pressure. Probably we do more of that in the operating room than is done anywhere else. One of the things that people need to keep in mind is that you *can*, obstruct the airway by doing that. If it's not working, don't! When applying a Sellick maneuver—if you can't ventilate with cricoid pressure application, try letting it go.

Kaye: A little homily. Our goal is to take basic science and translate it into practice. The proper conduit is education. None of us became physicians, nurses, or RCPs by just grabbing a toy and playing with it. I think that if the scientists tell us that ventilation is or is not necessary or bag-mask ventilators are good or bad, it is up to us to translate that science into practice. The bag-mask ventilator is clearly the major culprit. Several years ago American Heart almost threw it out—it was no longer required for "certification" back in the old days. The fact is that everybody grabs that device. Everybody loves it! When I respond to

a hospital code, the first thing I do is tell the airway manager to tilt the head back, make sure there's a tight mask fit, and "Watch the chest rise!" The fact is it's not being used right. I think there is a role for the bag-mask ventilator. But I think our problem is that our educational process is clearly deficient—let's not throw out a device because it's hard to teach. Let's make our teaching better so that it's effective.

Pepe: Would you like me to address the 5 points....

Branson: Well, if you'd like to.

Pepe: I can address them one by one if you like.

Branson: Are we talking about the controversies?

Pepe: Correct.

Branson: Sure. One was cricoid pressure. Do you want to say anything about cricoid pressure?

Pepe: It's good in adults during bag-valve-mask ventilation and in intubated patients to occlude the esophagus and, therefore, to diminish the risk of reflux and enhance the passage of gas into the trachea.

Branson: OK.

Pepe: If you do it right, teach it right, with the proper caveats included, it's not that hard to do, and you usually don't compress the airway. You could, conceivably, press too hard, but generally it's the reasonable thing to do. OK?

Branson: The second was Do we have to use a tidal volume of 0.8 to 1.2 liters?

Pepe: OK. First of all, when you stop breathing you don't sigh anymore. When you're breathing normally,

you sigh normally, right? However, if you stay at the same normal tidal volume of 400 and 500 mL without sighs over a period of time, you would probably have airway closure in dependent lung zones with resulting shunt and hypoxemia.¹ So, what we've done on ventilators is to put people on at 8 mL/kg or greater, generally at an initial rate of 10-12, and we've found that from our collective experience that the lung deflation is avoided. This becomes particularly important during CPR when you're pressing on the chest. Lung compliance is already going down anyway because by the time you intubate the patient. Even if you haven't yet started CPR, the patient's lung volume has already begun to deflate to some extent.¹ Therefore, the key is to maintain adequate lung inflation. Now what you're getting at is also key—What's the rate at which we deliver this adequate lung inflation? That's a little tougher to determine. On the ACLS committee, I would probably have voted for more like 5-6/minute rather than 10-12 breaths/minute during circulatory arrest because I think you need only a minimum number of breaths to maintain adequate lung inflation (which is probably at least 5 or 6 breaths). Unfortunately, most clinicians have been told in the past that they should be hyperventilating the patient at 12-15 breaths/minute. Therefore, 10-12 per minute was kind of a consensus compromise that came out of the panel's deliberations.² The next question is, Why apply BLS standards to ALS devices? In other words, the rate of delivery of air into the intubated person (ie, over a 1-2 second period)? The rationale that distribution of gas actually is better at the lower inspiratory rate with positive pressure breaths (vs taking a spontaneous breath). A rapidly delivered positive-pressure breath may not distribute optimally. I think it's a small issue, however. I

think that once you've intubated them, it's not that big a deal about how fast you deliver the gas—Just how often and how deep.

1. Pepe PE, Copass MK, Joyce TH. Prehospital endotracheal intubation—rationale for training emergency medical personnel. *Ann Emerg Med* 1985;14:1085-1092.
2. American Heart Association. Adult Advanced Cardiac Life Support (ACLS). Part III, Guidelines for Cardiopulmonary Resuscitation and Emergency Care. *JAMA* 1992;268(10-28-92):2199-2241.

Branson: It's not an issue at all, once they're intubated. That was why I was curious.

Pepe: Well, it is a physiologic issue, but it's probably clinically unimportant. So, I agree with your conclusion. As far as I'm concerned, once they're tubed, if you maintain adequate inflation by at least a few breaths per minute, you're probably OK during circulatory arrest. The guidelines were influenced by some of the participating physiologists. As a clinician as well as a pulmonary physiologist I'm not inflexible about that consensus guideline. Next issue: Is ventilation necessary at all? Probably. But, it's probably not as necessary as we thought in the past.¹

1. Pepe PE, Copass MK, Warnke WJ. Emergency medical services and systems of out-of-hospital resuscitation. In: *Cardiac Arrest: The pathophysiology and therapy of sudden death*. Paradis, Nowak, Halperin, eds. Baltimore: Williams & Wilkins, in press.

Branson: I agree with you, but, again, the people who are doing ARDS research, as you're probably aware, are getting away from even 10 mL/kg. They're doing 6 mL/kg tidal volume on patients with ARDS. Again, different lung pathologies.

Pepe: Aren't these ARDS people on significant levels of PEEP?

Branson: Yes.

Pepe: So it's still an adequate lung-inflation issue, to some extent.

Branson: Sure.

Halperin: Another thing peculiar to Johns Hopkins—that's really where the rate of 10-12, and minute volume of 0.8-1.2, came from. It's buried in the CPR literature. It was done by Peter Safar in the late 50s and early 60s. He actually recruited volunteers and did mouth-to-mouth ventilation on them and lots of other kinds of ventilation, and he found that it took that amount of ventilation to maintain their blood gases. That's where the ABCs came from—because he added airway and breathing to chest compression. That work has been criticized a lot because, clearly, the heart was beating, and probably a lot more ventilation was needed. However, he still defends it. The other thing is that Idris² and his group down in Gainesville have done some work looking at how much ventilation is necessary. Their work was in pigs. They haven't extended it to patients yet—but they looked at various gas mixtures to try to simulate the low oxygen and high CO₂ of mouth-to-mouth ventilation. What they found was that in animals that were hypercarbic there was actually a substantial decrease in survival, compared with animals that were not hypercarbic. The implication is that you need enough ventilation to prevent hypercarbia, but, again, those studies are very preliminary.

2. Idris A, Fuerst R, Wenzel V, Becker L, Orban D, Banner M. Does hypoxia or hypercarbic acidosis independently affect survival from cardiac arrest? (abstract) *Circulation* 1993;88(4, Part 2):1225.

Aufferheide: To comment and echo some previous comments—unlike ACLS, BLS has a lot fewer data on which to base its recommendations, and there's probably no other aspect of BLS that is in need of research more than this one. We're hoping that in the next 5 years there will be a lot more research, so that more intelligent recommendations can be made, both for barrier devices and for airway adjuncts.

Kaye: I will second that. I think we have to remember that CPR probably would rate about a 1Ib. I'm not sure about a 1II, but it's probably a 1Ib. And it's all based on poor data from the past, beautifully amplified by BLS practitioners on recording CPR mannequins and the committees who sat in camera late at night figuring out guidelines. I don't mean to be critical, but it's just a fact. I think that absolutely, as they say, more work is needed. Basic science needs to critically evaluate every one of these sacred cows and put them to rest. I think it's very important because of the question Where did it all come from?² I'm not sure—some science, some tradition.

Barnes: I have a lot of trouble with the work that Banner and Melker did¹ because when you look at their data at low compliances, then the difference between gas to the stomach and gas to the lungs really comes together. I think it really behooves us, for unintubated patients, to look and see what their lung compliance is. We may

have someone who went into pulmonary edema and then arrested, whose lungs are full of water, who really needs to be treated differently. Maybe the patient needs to be intubated sooner. I think certain patients need to be intubated earlier. Bag-valve-mask is not the end-all. Some people think as long as you do bag-valve-mask properly, you can take as much time as you want to intubate, but with some patients, no matter how well you use the resuscitator, you're going to put a lot of air in the stomach.

1. Melker RJ, Banner MJ. Ventilation during CPR: two-rescuer standards reappraised. *Ann Emerg Med* 1985;14:397-402.

Branson: Because we're talking about in-hospital resuscitation, ideally, everybody ought to be intubated as soon as the equipment arrives. For most of us, I believe, this occurs in fairly short order. If we change the setting to ventilation provided by a BLS provider then the issue of lung compliance and gastric insufflation is more important.

Barnes: There are some papers that have shown in simulated cardiac arrests that the nursing staff doesn't even get the oxygen flowing before the resuscitation team arrives, so the first responders are further off the mark than we think they are on the basic BLS concepts.

1. Safar P. Ventilatory efficacy of mouth-to-mouth artificial respiration: airway obstruction during manual and mouth-to-mouth artificial respiration. *JAMA* 1958;167:335-341.

1. Palmisano JM, Akingbola OA, Moler FW, Custer JR. Simulated pediatric cardiopulmonary resuscitation: initial events and response times of a hospital arrest team. *Respir Care* 1994;39:725-729.
2. Sullivan MJJ, Gorden GH. Simulated cardiac arrests for monitoring quality of in-hospital resuscitations. *Lancet* 1986;1:618-620.

Methods of Emergency Drug Administration

Dean Hess PhD RRT and Ravikumar Alagar MD

- I. Introduction
- II. Intratracheal Drug Administration
- III. Intraosseous Drug Infusion
- IV. Vascular Access
- V. Intracardiac Drug Administration
- VI. Role of the Respiratory Therapist

Introduction

Administration of drugs is an important adjunct to successful resuscitation. Other than oxygen, administration of drugs is secondary to basic life support, defibrillation, and airway management. During cardiopulmonary resuscitation (CPR), drugs can be administered by central or peripheral infusion, intracardiac injection, endotracheal instillation, or intraosseous infusion.

Intratracheal Drug Administration

Intratracheal drug administration during CPR is appealing for several reasons. The airway is often more easily secured than is vascular access—particularly in pediatric patients, obese patients, dialysis patients, intravenous narcotic abusers, and patients with profound peripheral vascular collapse. Drugs absorbed from the airway return to the left atrium and should, therefore, be delivered quickly into the systemic circulation.¹ Although this route of emergency drug administration has been widely accepted and frequently used, it has undergone little scientific investigation on human subjects in cardiac arrest.¹⁻³

Initial interest in endotracheal drug administration occurred after data by Redding et al⁴ were published.

Dr Hess is Instructor in Anesthesia, Harvard Medical School and Assistant Director, Department of Respiratory Care, and Dr Alagar is Senior Fellow in Pulmonary and Critical Care Medicine—Massachusetts General Hospital, Boston, Massachusetts.

A version of this paper was presented by Dr Hess during the Journal Conference, "Resuscitation in Acute Care Hospitals" held in Cancun, Quintana Roo, Mexico in October 1994.

Reprints: Dean Hess PhD RRT, Department of Respiratory Care, Ellison 401, Massachusetts General Hospital, Boston MA 02114

Effectiveness of epinephrine administration (1 mg) using 8 methods (n=10 in each group) was evaluated in mongrel dogs (Fig. 1). Intratracheal administration of epinephrine (1 mL 1:1,000, diluted to 10 mL total volume) was as effective as intravenous (I.V.) or intracardiac administration.

AHA Recommendations

The American Heart Association (AHA)⁵ recommends endotracheal drug administration of epinephrine, lidocaine, and atropine if venous access is delayed. The recommended intratracheal dose is 2-2.5 times the I.V. dose, diluted to a volume of 10 mL in water or normal saline (NS). The AHA recommends that a catheter be passed beyond the endotracheal tube, chest compressions interrupted, drug injected through the catheter, several quick insufflations given, and chest compressions resumed.

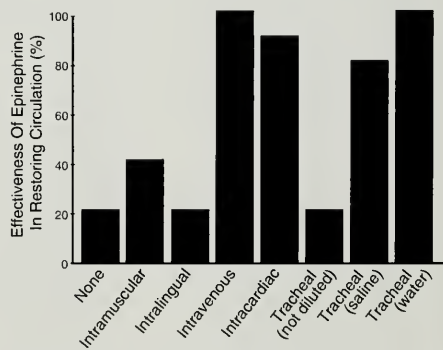


Fig 1. Effectiveness of epinephrine in restoring circulation in cardiac arrested dogs by route of administration; n = 10 in each group. (From Reference 4, with permission).

Drugs Administered by This Route

Epinephrine has been the drug most commonly administered by the tracheal route.⁶⁻¹⁸ However, the tracheal administration of other drugs has been described—lidocaine,¹⁹⁻²⁵ atropine,^{26,27} diazepam,^{28,29} and naloxone.³⁰ Because the tracheal route is attractive for drug administration, it is likely that other drugs may be administered by this route in the future.^{31,32} A requirement for tracheal drug administration is that it be effective at a dose that can be delivered in a relatively small volume (eg, ≤ 10 mL). With larger drug volumes, the potential for interference with gas exchange is greater, although this has not been scientifically evaluated. Even though emergency tracheal administration of a variety of drugs has been described, it must be understood that the scientific basis for the routine use of this technique has not been well-established in patients.^{2,3}

Catheter vs Direct Instillation—Effect of Hyperinflation

As stated previously, the AHA has recommended that a catheter (eg, pediatric feeding tube) be passed through the endotracheal tube for administration of emergency drugs. The AHA also recommends that chest compressions be stopped during this procedure, and that several quick insufflations should be given following drug instillation.⁵ The rationale for this is that the use of a catheter prevents adherence of the drug to the endotracheal tube wall. This is a controversial topic and both the catheter-instillation and direct-instillation methods have their proponents.

There is little support in the literature for the catheter-instillation technique. Ralston et al¹⁰ studied intrapul-

monary epinephrine administration in a group of dogs in ventricular-fibrillation cardiac arrest, and compared those to a group that did not receive epinephrine by any route. Intrapulmonary epinephrine administration was achieved by use of a Swan-Ganz catheter, which was passed with its balloon inflated to a wedge position deep in the bronchial tree. Compared to the group that received no epinephrine, those who received intrapulmonary epinephrine had greater tissue blood flow to the heart, brain, kidneys, and adrenal glands (Fig. 2). However, there are several shortcomings of this study—direct instillation was not compared with the catheter technique, and intrapulmonary drug administration was not compared to other routes of epinephrine administration. In a dog study, Mazkereth et al¹⁶ found higher plasma epinephrine levels with peripheral bronchial instillation (using a 16-gauge 24-inch catheter) compared to direct endotracheal instillation (Fig. 3). However, physiologic response (heart rate and mean arterial pressure) were similar with both approaches. They speculated that some portion of endotracheally instilled drugs may remain in the endotracheal tube and large conducting airways, preventing effective absorption.

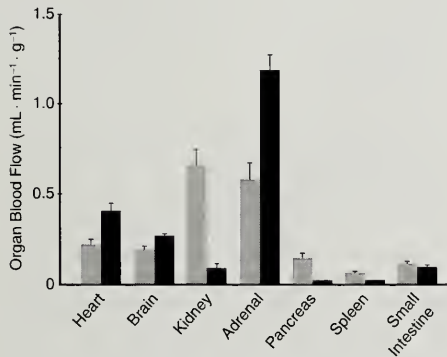


Fig 2. Tissue blood flow during 10-20 minutes of CPR to seven tissue beds. Group I is control (□) and Group II (■) received intrapulmonary epinephrine. (From Reference 10, with permission).

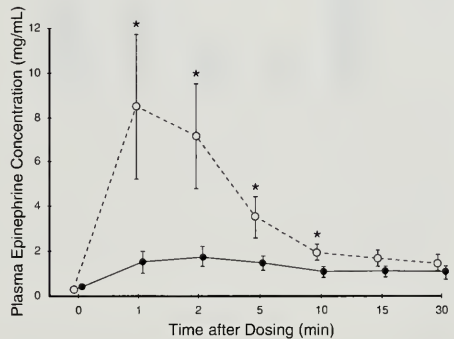


Fig 3. Time course of plasma epinephrine concentration after endotracheal (ET —○—) and peripheral bronchial (PB —●—) administration; symbols are mean $\pm$ SD. * $p < 0.05$ between the 2 routes. (From Reference 16, with permission).

Mace²¹ compared 5 methods of intratracheal administration of lidocaine in a canine model. All of the dogs were healthy (none were in cardiac arrest). Each dog received a bolus of endotracheal lidocaine (3 mL) using 1 of 5 techniques, and plasma lidocaine levels were drawn at 15, 30, and 60 minutes. The 5 techniques used were injection of lidocaine directly from a syringe into the endotracheal tube, injection of lidocaine directly into the endotracheal tube through a 13-gauge spinal needle, dilution of lidocaine to a volume of 6 mL before instillation, injection of lidocaine followed by instillation of NS of an equal volume, and in-

jection of lidocaine through a catheter extending beyond the tip of the endotracheal tube (size and type unspecified). With undiluted drug (3 mL), plasma lidocaine levels were initially greater with catheter instillation than with direct instillation (Fig. 4A), but this difference disappeared by 15 minutes following administration (Fig. 4B). Interestingly, Mace also found that the greatest plasma lidocaine levels occurred when the drug was diluted to 6 mL and directly instilled into the endotracheal tube (Fig. 4).²¹

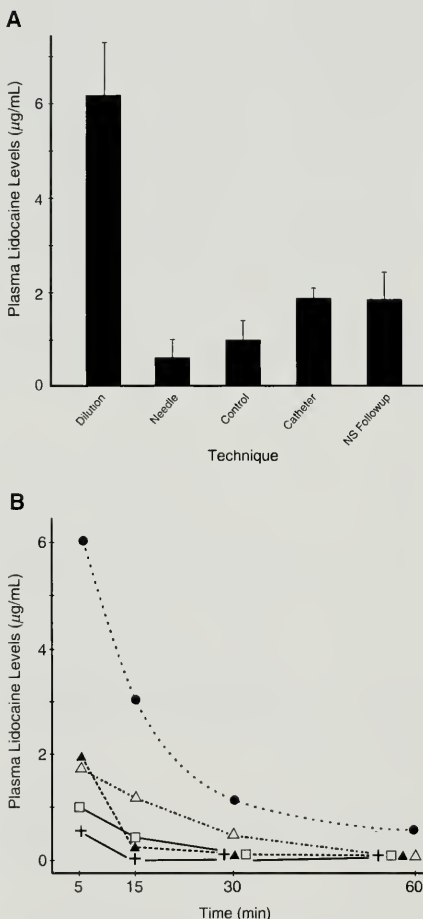


Fig 4. (A) Mean $\pm$ SD plasma lidocaine level for 5 techniques of endotracheal administration at 5 minutes after administration. (B) Mean plasma lidocaine levels as a function of time for 5 techniques of endotracheal administration— $\bullet$ dilution, $\square$ control, $+$ needle, $\blacktriangle$ catheter and $\triangle$ NS follow up. (From Reference 21, with permission).

Several comparisons of instillation through a catheter versus direct instillation have shown no benefit of instillation through a catheter.^{18,22,24,25,33} The lack of superiority of catheter instillation has been shown using contrast material,³³ lidocaine,^{22,24,25} and epinephrine.¹⁸ Greenberg and Spivey³³ used contrast material to compare 4 methods of endotracheal drug administration—direct instillation followed by 5 rapid insufflations, instillation through a catheter followed by 5 rapid insufflations, direct instillation but no rapid insufflations, and instillation through a catheter but no rapid insufflations. They found that manual ventilation produced bilateral distribution of the contrast, but use of catheter instillation offered no benefit (Table 1).³³ In several studies using anesthetized patients,^{24,25} Prengel et al evaluated lidocaine blood levels following direct endotracheal instillation or deep endobronchial instillation using a bronchoscope (10 mL at 2 mg/kg in both groups). In one study, it was found that therapeutic blood levels could be achieved without toxic blood levels with either approach.²⁴ In the other study, it was shown that initial lidocaine blood levels were higher with direct endotracheal tube instillation (Fig. 5).²⁵

Table 1. Effect of Method of Instillation on Distribution Pattern and Depth of Penetration.*

Method	Bilateral Distribution (%)	Penetration to Terminal Bronchioles (%)
Direct instillation and manual ventilation following instillation	100	67
Catheter instillation and manual ventilation following instillation	100	83
Direct instillation and no manual ventilation following instillation	0	17
Catheter instillation and no manual ventilation following instillation	0	17

*From Reference 33, with permission.

Using swine in whom cardiac arrest had been induced, Jasani et al¹⁸ compared 3 methods of radioactive epinephrine administration (5 mL in each group)—direct instillation into the endotracheal tube, injection via a feeding catheter, and injection via the monitoring lumen built into the sidewall of the endotracheal tube. They documented no difference in successful resuscitation between the 3 groups and found that little epinephrine adhered to the en-

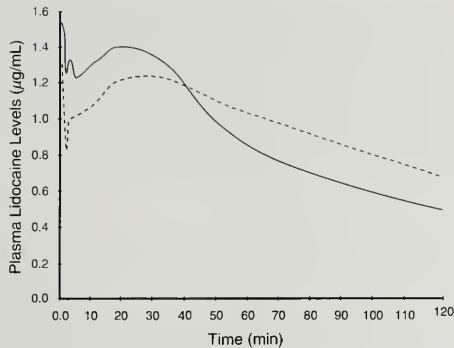


Fig 5. Plasma lidocaine concentrations for the first 2 hours after 2.0 mg/kg administered by direct endotracheal instillation (—) or intrasegmental instillation (---). (From Reference 25, with permission).

dotracheal tube wall ($\leq 1.5\%$). They also found that bilateral distribution of the epinephrine occurred in about 40% of the animals (no difference between groups), and distribution pattern did not affect successful resuscitation.

From our review of the literature, there appears to be no advantage of the catheter-instillation method over direct instillation. The catheter-instillation method is labor intensive, and requires interruption of ventilation and chest compressions. Direct instillation is quicker, less expensive, and easier to perform. Techniques that involve deep endotracheal instillation (eg, bronchoscopic instillation and instillation through a Swan-Ganz catheter wedged into the airway) are almost impossible to use during a cardiac arrest. We are also concerned that deep intrabronchial instillation in patients with lung disease could result in drug delivery to parts of the lung where uptake might be compromised. Following instillation with 5 rapid insufflations does seem reasonable, and most studies have used this technique.

Dose

Based upon early studies by Roberts et al,^{6,7} a common recommendation is to use an endotracheal dose of epinephrine that is ten times the intravenous dose. Using a pig model, Hörnchen et al¹³ found similar plasma concentrations and physiologic effects when intravenous epinephrine was compared to endotracheal administration at ten times the intravenous dose. Crespo et al¹⁵ compared 2 doses of endotracheal epinephrine (0.01 mg/kg and 0.1 mg/kg) in a swine model. The lower epinephrine dose produced plasma epinephrine levels that were not clinically different from control, whereas the higher dose produced significant increases in plasma epinephrine levels (Fig. 6). Although the dose of epinephrine should be increased for

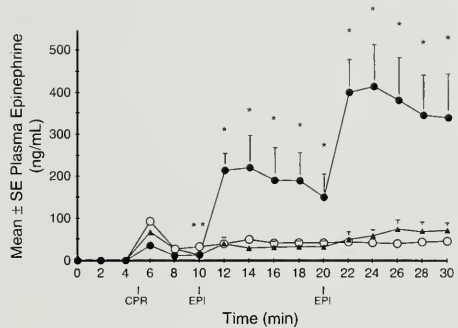


Fig 6. Mean plasma epinephrine levels as a function of time for the control (○), epinephrine 0.01 mg/kg (▲), and epinephrine 0.1 mg/kg (●). * indicates epinephrine 0.1 mg/kg significantly different from control and 0.01 mg/kg dose ($p < 0.05$). ** indicates that the control group is different from both epinephrine groups ($p < 0.05$). (From Reference 15, with permission).

endotracheal administration, whether this is necessary for other drugs such as lidocaine,^{24,25} diazepam,²⁹ and atropine²⁶ is unclear.

Diluent Volume

It is generally accepted that endotracheal drug administration requires dilution to a volume of 5-10 mL. Redding et al⁴ found that an endotracheal dose of 1 mL epinephrine was ineffective, whereas endotracheal epinephrine diluted to 10 mL was as effective as intravenous or intracardiac administration. Mace²¹ found that endotracheal instillation of lidocaine resulted in much greater plasma lidocaine levels when the drug was diluted to 6 mL before instillation.

Normal Saline vs Water Diluent

For endotracheal drug administration, there is a theoretical advantage to dilution with water rather than NS. Dilution with water should result in a lower osmolality than NS dilution, thus, promoting drug uptake from the pulmonary parenchyma. In dogs, Greenberg et al³⁴ found that administration of NS by the endotracheal route produced fewer detrimental effects on arterial blood-gas values than did the administration of distilled water. However, a dose of 2 mL/kg was used in that study, which is much greater than that typically used for endotracheal drug administration (eg, 140 mL for a 70 kg person). Redding et al⁴ found that successful resuscitation of dogs was virtually identical whether NS or water was used to dilute epinephrine, but the return of spontaneous circulation

was more rapid in the group who received water as the diluent. In anesthetized humans with normal cardiopulmonary function, Hähnel et al²² compared endotracheal administration of lidocaine diluted to 10 mL with distilled water and NS. At 5 minutes following instillation, plasma lidocaine levels were greater with the water-diluent group than the NS-diluent group. Further, the average drop in P_{aO_2} was greater in the NS-diluent group (60 torr) than the water-diluent group (40 torr). Although these few studies are not conclusive, it appears that drug dilution with water may be superior to NS dilution for endotracheal administration of epinephrine.

Depot Effect

Delayed absorption from the lung to the bloodstream of endotracheally administered drugs has been reported for epinephrine and lidocaine. This effect has been referred to as a depot effect. The depot effect was first described by Roberts et al,^{6,7} who reported rapid and sustained blood levels of epinephrine following endotracheal administration in anesthetized dogs. In anesthetized humans, Prengel et al²⁵ described a depot effect with endotracheal administration of lidocaine, in which drug absorption was found to be biphasic (Fig. 7).²⁵ A depot effect has also been demonstrated in animal models of cardiac arrest.^{13,15} Whether or not this depot effect is good or bad is a topic of controversy. With epinephrine, delayed clearance could lead to persistent tachycardia, hypertension, and dysrhythmias.

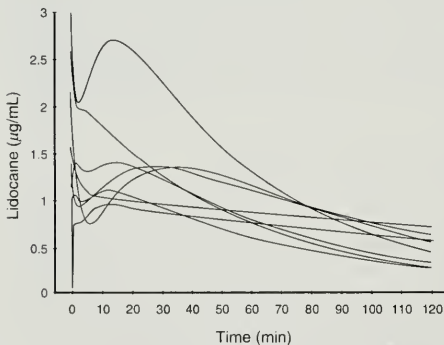


Fig 7. Curve fittings for plasma lidocaine concentrations of 8 patients after endotracheal administration. Note the biphasic pattern of absorption. (From Reference 25, with permission).

Effect on Pulmonary Gas Exchange

The administration of drugs by the endotracheal route is presumably safe, although this has not been thoroughly

evaluated. Hähnel et al²³ reported a decrease in P_{aO_2} following endotracheal administration of lidocaine. However, none of the patients in this study became hypoxemic secondary to the procedure due to supplemental oxygen administration. Two other studies, one in humans²² and one in dogs,¹⁶ reported no deleterious effect of endotracheal drug administration on arterial blood-gas values.

Animal vs Human Studies

Most studies of endotracheal drug administration have been conducted using animal models, and few studies have used human subjects. This is understandable because it is difficult to conduct randomized, blind, placebo-controlled clinical trials on humans who are in cardiac arrest, and because endotracheal drug administration is widely accepted and recommended by the AHA. The first report of successful use of endotracheally administered epinephrine to a patient in cardiac arrest was that of a 13-day-old boy.⁹ In humans, reports on the use of endotracheal drug administration have been primarily anecdotal.

McCririck and Kestin¹⁷ compared systolic blood pressure responses to intravenous and endotracheal epinephrine administration in 12 patients undergoing elective hip replacement. Intravenous epinephrine was administered as 0.1 µg/kg diluted to 5 mL with NS. The dose of endotracheal epinephrine was 0.5 µg/kg diluted to 5 mL with NS, and administered through a fine-bore catheter positioned 1 cm beyond the tip of the endotracheal tube. Tracheal administration was followed by 6 large insufflations. Tracheal administration of epinephrine failed to produce a clinically important increase in systolic arterial blood pressure in any patient (Fig. 8), suggesting that a tracheal dose > 5 times the intravenous dose may be neces-

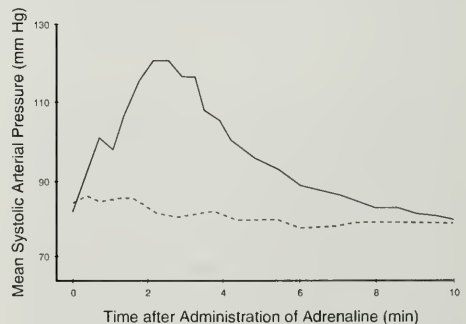


Fig 8. Mean systolic arterial pressure after intravenous (0.1 µg/kg, —) or tracheal (0.5 µg/kg, ----) epinephrine in 12 patients undergoing elective surgery. (From Reference 17, with permission).

sary to produce a physiologic response. A limitation of this study was that none of the patients were in cardiac arrest, and the results of the same study on patients in cardiac arrest might have been different.

Quinton et al¹⁴ compared endotracheal and intravenous epinephrine in 12 patients who presented in the emergency department in cardiac arrest. Patients were randomized to receive either intravenous epinephrine (1 mL 1:1,000 in antecubital vein, $n = 7$) or endotracheal epinephrine (10 mL 1:1,000 directly instilled into the endotracheal tube followed by 6 insufflations, $n = 5$). Virtually no change occurred in arterial epinephrine levels in patients who received endotracheal epinephrine, whereas there was a significant increase in arterial epinephrine levels in patients who received intravenous epinephrine (Fig. 9). Although the sample size was small, the authors concluded that the endotracheal route is not reliable for epinephrine administration in patients with out-of-hospital cardiac arrest.

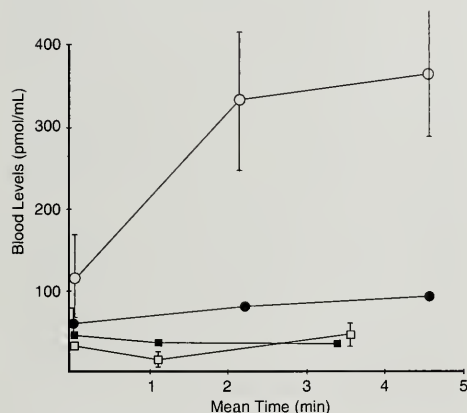


Fig 9. Arterial epinephrine (adrenaline, ●) and norepinephrine (noradrenaline, ○) levels before and after epinephrine administration by intravenous route (●, ○) and intratracheal route (■, □) in 12 cardiac-arrested patients. (From Reference 14, with permission).

Recommendations

We believe that endotracheal administration of some drugs (ie, epinephrine, lidocaine, atropine) may be effective. Our recommendations for use of this method, based upon our review of the literature, are listed in Table 2. One must recognize that very little research using endotracheal drug administration has been done on humans in cardiac arrest. However, it is difficult to conduct randomized, blinded clinical trials in such patients. Although data in animal models and human subjects not in cardiac arrest

support the use of this method, studies on patients who are actually in cardiac arrest have not uniformly shown that tracheal instillation is an effective method of drug delivery during resuscitation.

Table 2. Endotracheal Drug Administration during Cardiac Arrest: Recommendations.

- Endotracheal drug administration should not be considered a first line therapy during cardiac arrest; this route should only be used if venous access cannot be secured.
- At least for epinephrine, an appropriate endotracheal dose is 10 times the intravenous dose.
- No clear superiority has been shown for instillation through a catheter, and, therefore, direct instillation into the endotracheal tube should be used.
- The dose should be diluted to 10 mL with either normal saline or water.
- Five rapid insufflations should be used after endotracheal drug administration.

Intraosseous Drug Infusion

Intraosseous infusion as a technique for venous access was described by Drinker in 1922.³⁵ During the 1940s, experience with this technique was described by many authors as summarized by Rosetti and colleagues (Table 3).³⁶ Tocantins³⁷ may have been the best-known advocate for intraosseous infusion in the 1940s. In the 1950s and 1960s, intravenous catheters and techniques were developed and improved, and intraosseous infusion was largely forgotten. However, in the past 10 years, there has been renewed interest in the use of intraosseous infusion,³⁸⁻⁴¹ particularly in patients in whom emergency intravenous access is difficult. The study by Rosetti et al³⁶ reported that venous access required more than 10 minutes in 24% of patients in cardiac arrest in the pediatric emergency department, and intravenous access was never achieved in 6% of arrests. Kruse et al⁴² suggested that some features of intraosseous infusions are attractive: (1) cannulation can be performed in ≤ 30 s, (2) the procedure has a high success rate, even in the hands of paramedic personnel, (3) the anatomic landmarks used to facilitate catheter placement are easily identified, (4) the complication rate is low, and (5) a wide variety of drugs and fluids can be administered by this route.

Intraosseous infusion uses the rich vascular network of long bones to transport fluids and drugs to the venous circulation.⁴⁰ The medullary cavity is composed of a spongy network of venous sinusoids that drain into a central venous canal, and from there into the venous circulation (Fig. 10). The intraosseous route has been used for administration of numerous drugs and solutions,³⁸⁻⁴⁵ including NS, glucose, blood, sodium bicarbonate, atropine, dopamine, epinephrine, diazepam, antibiotics, phenytoin,

Table 3. Studies of Intraosseous Infusion Conducted in the 1940s.*

Author (Date)	Number Successful Infusions	Number Failed Infusions%	Complications Noted
Arbeiter (1944)	35	8 (18.66)	0
Bailey (1944)	60	?	1 Posterior sternal perforation
Behr (1944)	60	?	1 Osteomyelitis
Bielstein (1944)	58	?	0
Clement (1947)	65	?	3 Osteomyelitis
Couder (1948)	100+	?	0
Dardinski (1945)	?	?	1 Mediastinitis
Ellison (1944)	40	?	0
Elston (1947)	97	15 (13.4)	1 Subcutaneous abscess
Giraud (1945)	20	?	0
Gunz (1945)	35	1 (2.9)	3 Osteomyelitis
Heinild (1947)	982	18 (1.8)	5 Osteomyelitis
Heyrouti (1947)	310	?	0
Higgins (1947)	120	?	0
Massey (1950)	72	12 (14.3)	1 Osteomyelitis
Meela (1944)	326	0 (0.0)	1 Arterial thrombosis
O'Neill (1942)	116	?	1 Posterior sternal perforation
Pillar (1954)	35	?	1 Subcutaneous sloughing 2° level
Quilligan (1946)	45	?	1
Ravitch (1943)	?	?	1 Anterior mediastinitis
Reich (1945)	35	?	0
Reisman (1944)	?	?	1 Subcutaneous abscess
Romero (1945)	77	?	0
Rooney (1944)	100	2 (2.0)	2 Osteomyelitis
Sondergaard (1946)	750	?	5 Osteomyelitis
Sutton (1947)	200	?	0
Texter (1948)	300	25 (7.7)	2 Osteomyelitis, 1 subcutaneous abscess
Tocantins (1943)	79	9 (10.2)	2 Osteomyelitis, 1 subcutaneous abscess
Valdes (1977)	15	2 (11.8)	0
Williams (1948)	138	?	1 Osteomyelitis

* ? = information not available (From Reference 36, with permission)

succinylcholine, lidocaine, crystalloids, dextran, isoproterenol, pancuronium, bretylium, and contrast material. Drug doses and fluid-administration rate by this route are the same as the intravenous route.⁴² In addition to an infusion site, intraosseous samples have been evaluated for their use as a source of blood for laboratory studies.⁴⁶

AHA Recommendations

The AHA⁵ states that intraosseous infusion is an excellent alternative when intravenous access is not available, particularly in pediatric patients. The AHA recommends that the intraosseous dose be the same as the intravenous dose, although a higher dose may be needed for intraosseous epinephrine.

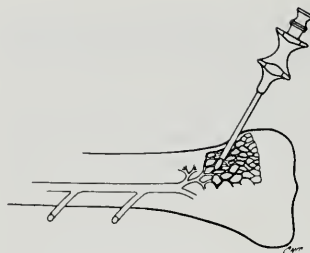


Fig 10. Intraosseous venous system demonstrates position of intraosseous needle in the medullary sinusoids. (From Reference 40, with permission).

Technique

Sites for intraosseous infusions include the sternum, humerus, clavicle, femur, and tibia.³⁸⁻⁴² Of these, the tibia and femur are most commonly used. The sternum, humerus, and clavicle cannot be used during cardiopulmonary resuscitation. In children less than 6 years of age, the proximal tibia is most frequently used because it has a broad flat surface with a thin layer of skin covering the bone. The distal tibia is also a useful site, and can be used in children or adults. The distal femur is usually not a first choice among sites because overlying muscles and fat make palpation of landmarks difficult.

The insertion site in the proximal tibia is shown in Figure 11. The tibial tuberosity and medial border of the tibia are palpated. Halfway between these points (ie, on the flat medial surface of the proximal shaft) and 1-2 cm distally, the needle is inserted in a caudal direction. The optimal insertion site for the distal tibia (Fig. 12) is the medial surface proximal to the medial malleolus, and the needle is inserted in a cephalad direction.



Fig 11. Insertion site in the proximal tibia for intraosseous needle. The tibial tuberosity and medial border of the tibia are palpated. Halfway between these points and 1-2 cm distally, the needle is inserted pointing away from the joint space, in a caudal direction. (From Reference 40, with permission).

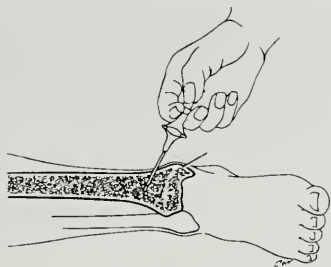


Fig 12. Insertion site for intraosseous needle in the distal tibia. The needle is inserted in the medial surface of the distal tibia at the junction of the malleolus and shaft of the tibia. It is inserted away from the joint space, in a cephalad direction. (From Reference 40, with permission).

Any needle with a stylet can be used.³⁸⁻⁴¹ However, a hollow needle may become plugged with bone. In children <18 months of age, an 18 or 20-gauge spinal needle can be used. In older children, a 13- to 16-gauge bone marrow needle can be used. There are also several commercially available intraosseous needles designed especially for this procedure. The skin should be cleansed with an antiseptic (iodine or alcohol), and a local anesthetic should be used if the patient is conscious. The needle is inserted perpendicular to the bone, with the bevel pointing away from the joint. A rotary motion is applied until there is a slight decrease in resistance ('*trap-door effect*' or '*soft pop*'), indicating that the cortex of the bone has been punctured. Care must be taken to avoid advancing the needle through the opposite side of the bone. To confirm the position of the needle, a syringe is attached and blood or marrow is aspirated.

The technique of intraosseous infusion has been successfully incorporated into the prehospital practice of paramedics.⁴⁷⁻⁴⁹ Paramedical personnel can be taught this technique in a relatively short training session. Fresh chicken thigh bones or pig shin bones are commonly used for training and simulations.

Complications

In general, the complication rate for this technique is low.³⁸⁻⁴¹ The most common complication is inability to enter the bone marrow cavity. Extravasation of fluid can occur around the puncture site, or from fracture sites and holes in the cortex from unsuccessful attempts. For this reason, intraosseous infusion is not recommended in a fractured bone or in one with an unsuccessful attempt. Multiple attempts at intraosseous infusions can significantly decrease vascular drug levels, presumably due to extravasation.⁵⁰

Osteomyelitis has been a concern with intraosseous infusion. However, the risk of this is actually low (<1%).

Damage to the epiphyseal plate is a concern, but there has never been a reported case of this related to intraosseous infusion. Significant complications may occur with the sternal site, but this approach is virtually never used. Localized cellulitis and subcutaneous abscesses can occur (0.7%), but their incidence is probably not greater than that of localized infections with vascular catheters (3.7%). Although the complication rate is low, intraosseous infusions should be discontinued as soon as vascular access is secured.

Intraosseous Infusion in Adults

The intraosseous route for infusion has been used most frequently in children. The highly vascular red marrow of long bones is replaced with less-vascular yellow marrow at about 5 years of age.⁴² This yellow marrow still contains numerous venous sinusoids and allows modest infusion rates when cannulated. The site of infusion in adults is the distal tibia. Valdes⁵¹ described his experience with intraosseous infusion in 15 adults with a median age of 45 years. The patients, none of whom were in cardiac arrest, received infusions of 2-42 L over a period of 3.5-30 days. No serious complications occurred. Iserson⁵² described his experience with intraosseous infusion in 22 adults (age 36-84 years) in cardiac arrest. Temporally related pharmacologic effects were observed after intraosseous infusions of sodium bicarbonate, lidocaine, atropine, and vasopressors. There has generally been less interest in the use of intraosseous infusions in adults, perhaps because failure to establish vascular access is less common in adults than in children.

Infusion Rates

Several groups of investigators have evaluated infusion flowrates in animal models.⁵³⁻⁵⁵ Shoor et al⁵³ used a calf model (to simulate adult human size) and cannulated the right medial malleolus with a 13-gauge trocar. NS was infused at atmospheric pressure (bag suspended at 81 cm), 100, 200, and 300 torr. They found that increasing the pressure on the bag significantly increased the infusion flow (Fig. 13), with flows of about 2.5 L/h at 300 torr.

Hodge et al⁵⁴ used a dog model (4-6 kg puppies) and cannulated the proximal tibia. Infusion flowrates were determined using 20-gauge and 13-gauge needles at ambient pressure (bag hanging at 100 cm) and 300 torr. They found that flow in-vivo was much less than that during in-vitro conditions. Although flowrates were greater with pressure, there was no clinically important difference between 13-gauge and 20-gauge needles. They concluded that infusion rates with intraosseous infusions were inferior to that with central venous catheters (Fig. 14).

In a swine model, Schoffstall et al⁵⁵ compared intraosseous infusion rates with large and small animals, 13-

gauge and 20-gauge needles, NS and blood infusion, and gravity and pressure infusion. They found that infusion rate was affected by the size of the animal, pressure infusion, and whether NS or blood was infused (Figs. 15, 16).

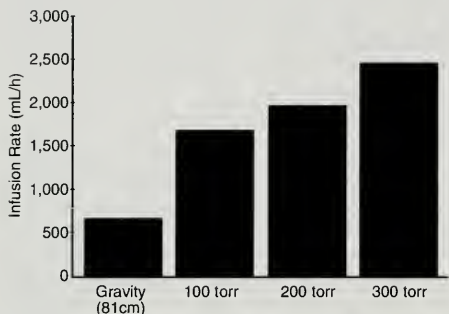


Fig 13. Intrasosseous infusion rates through 13-gauge needle inserted at the right tibial malleolus with gravity flow, 100, 200, and 300 torr infusion pressure. (From Reference 53, with permission).

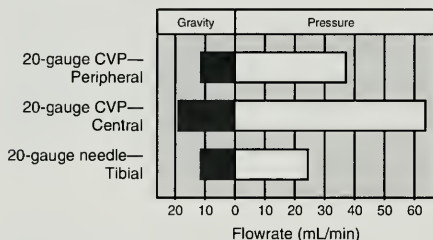


Fig 14. Comparison of flow rates in-vivo for different vascular sites. (From Reference 54, with permission).

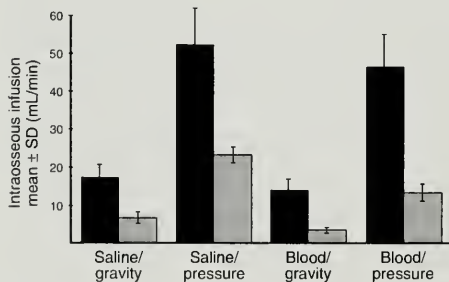


Fig 15. Rates of fluid administration in large swine at 100 cm H₂O (gravity) and 300 torr (pressure) through a 13-gauge (■) and an 18-gauge (□) catheter. (From Reference 55, with permission).

It is interesting that needle size was important in large animals, but not small animals. These results suggest that the rate limiting factor is not the resistance of the needle and tubing (as is the case with intravenous infusions), but rather the characteristics of the medullary space.

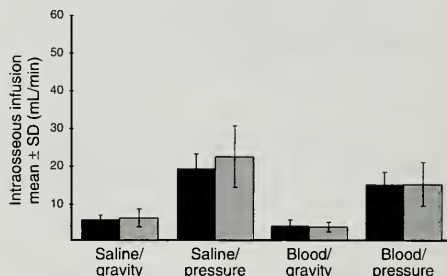


Fig 16. Rates of fluid administration in small swine at 100 cm H₂O (gravity) and 300 torr (pressure) through a 13-gauge (■) and an 18-gauge (□) catheter. (From Reference 55 with permission).

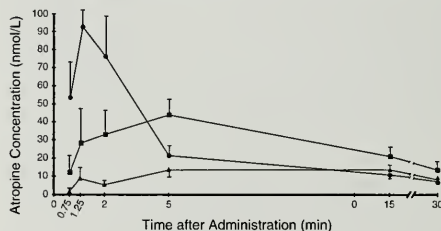


Fig 17. Plasma atropine concentrations (mean ± SE) in pigtail macaques at 6 time periods after 3 different routes of administration of 0.03 mg/kg atropine—intravenous (●, n = 5), intraosseous (■, n = 5) and endotracheal (▲, n = 6). (From Reference 27, with permission).

Comparisons of Intraosseous Infusions to Intravenous and Tracheal Drug Administration

The focus of several studies has been the comparison of intraosseous, intravenous, and/or intratracheal drug administration.^{27,56-59} Prete et al²⁷ compared plasma atropine concentrations in anesthetized monkeys via intravenous, endotracheal, and intraosseous administration. The same dose (0.03 mg/kg) was used in each case. Although initial plasma levels were greater with intravenous administration, the plasma concentration was actually greater with the intraosseous route than the intravenous route at 5 minutes (Fig. 17). The prolonged plasma concentrations with the intraosseous route also suggest a depot effect.

Several studies used radioactive isotopes to evaluate

peripheral to central delivery times with intravenous and intraosseous infusion.^{57,58} Generally, there was little difference between intraosseous infusion and peripheral venous infusion with regards to delivery of drug to the central circulation. Spivey et al⁵⁹ compare intraosseous, central, and peripheral routes of sodium bicarbonate administration during the resuscitation of dogs. They found that pH changes were virtually identical with both central-venous and intraosseous infusions of bicarbonate (Fig. 18).

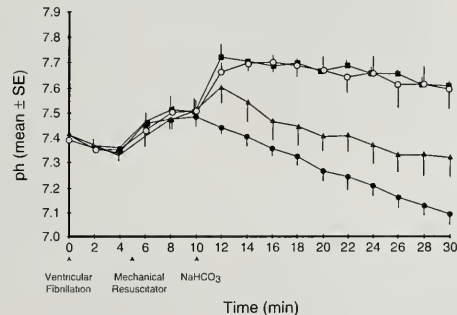


Fig 18. Effect of sodium bicarbonate on pH of arterial blood with 4 administration sites—control (●), peripheral (▲), central (■), intraosseous (○). Note that pH is similar with central and intraosseous infusion. (From Reference 59, with permission).

Recommendations

Intraosseous infusion should not be the technique of first choice for emergency drug administration. However, it should be considered in children ≤ 5 years of age if vascular access cannot be readily established and if the equipment is available for intraosseous infusion. The medication dose used with intraosseous infusion should be the same as that used with intravenous infusions. Intraosseous infusion can be used with adults, but only after other routes of drug administration (ie, intratracheal, central-vein) have failed. Intraosseous infusion can be used for continuous infusion, which makes it superior to tracheal drug instillation. The infusion rate can be increased by pressurizing the drug administration set. Paramedic personnel can be taught to successfully use intraosseous infusion. The intraosseous infusion should be considered short term, and the needle should be removed as soon as vascular access is secured.

Vascular Access

Vascular access for the administration of fluids and medications is an integral part of successful cardiopul-

monary resuscitation. Many techniques are available to achieve this, and each has its own merits and limitations. Many drugs may be administered intramuscularly or subcutaneously. However, absorption with these methods is often erratic and is dependent on the blood flow to these sites.⁶⁰ In cardiopulmonary emergencies, there is a low-flow state that leads to shunting of blood from skin and muscle, with impaired uptake of drugs and fluids from these sites, resulting in inadequate resuscitation. Whether vascular access is gained peripherally or centrally, proper technique must be followed. However, central venous access should be limited to operators who have been trained in the technique and are familiar with its problems.

AHA Recommendations

Administration of medications is secondary to other interventions such as basic CPR, defibrillation, and airway maintenance during resuscitation. If no vein has been cannulated before the arrest, a peripheral vein is the first choice. Peripheral veins commonly used for vascular access during resuscitation include the arm veins (dorsum of hand, antecubital, and cephalic), leg veins (femoral and saphenous), and external jugular veins. Peripheral-vein cannulation remains the vascular-access procedure of choice during cardiopulmonary resuscitation because it is safe, fast, and easy to perform.⁶¹ Resuscitation does not need to be interrupted and these veins are easily accessible. Peripheral-vein cannulation is much simpler to learn than central-vein catheterization (particularly by nonphysicians) and fewer serious complications occur with this method.

Peripheral-Vein Access

The most common sites of peripheral-venous access are the arms and legs. More proximal sites, such as the antecubital veins, are preferable to distal sites found in the hand. A large-bore needle (16-gauge or larger) is required for adequate drug delivery during resuscitation. Any peripherally administered intravenous drug should be rapidly delivered by bolus injection and followed with a 20-50 mL fluid flush to ensure adequate delivery of the drug to the central circulation.

The principal disadvantage of peripheral venous therapy is that a vein may be difficult to locate in a state of circulatory collapse. Isotonic solutions are tolerated, but hypertonic solutions are irritating to the vascular wall and should not be used. The risk of phlebitis is greater when saphenous veins are used.⁶² Pharmacokinetic studies suggest that a prolonged interval is required for peripherally administered drugs to reach the central circulation.^{63,64}

Despite its location in the neck, the external jugular vein is considered a peripheral site. Rapid entry to the right heart

is achieved via this vessel, and it may be used to monitor central-venous pressure. In the cardiac-arrest setting, airway management techniques (ie, head tilt) can make it difficult to cannulate the external jugular vein, and with the head movement associated with endotracheal intubation, the potential for dislodgement of the jugular-vein catheter is increased.

Central-Vein Access

The central veins used for vascular access are the femoral, internal jugular, and subclavian veins.⁶⁵ These are used when peripheral sites are not available or when central circulatory access is needed for measurement of central venous pressure, right heart catheterization, and transvenous cardiac pacing. The anatomic location of the central veins is predictable and permits placement of large-bore catheters. The greater flow through these vessels permits infusion of hypertonic solutions that would irritate peripheral vessels. In a state of cardiovascular collapse, rapid cannulation may be achieved by placing the head in a dependent (Trendelenburg's) position. For central venous cannulation, visualization of veins is not required and hence access to the circulation is achieved even when peripheral sites are collapsed.

Central venous cannulation is not without problems, and clinician skill and experience is vital to success. Resuscitation usually needs to be interrupted when neck veins are used. The internal jugular and subclavian veins lie close to their counterpart arteries, the apical lung pleura, the bronchial/cervical nerves, lymphatic duct and trachea. Consequently, there is a significant risk of pneumothorax and inadvertent arterial puncture.⁶⁶ When the femoral vein is cannulated, a long catheter that extends above the diaphragm is required for rapid delivery of medications and fluids to the central circulation.⁶⁷ The advantages and disadvantages of femoral cannulation are listed in Table 4. Other problems associated with central venous cannulation include hydrothorax,⁶⁸ catheter kinking or knotting,⁶⁹ possible shearing of the vessel with guide wire manipulation, cardiac tamponade,⁷⁰ and arteriovenous fistula.⁷¹

Catheter Types

Catheter insertion can be achieved using 1 of 3 types of cannulae (Fig. 19⁶¹). Insertion site determines the length of the needle and cannula required. Shorter catheters are used to cannulate peripheral veins. When volume expansion is required, the highest flow is achieved with a short, large-gauge catheter.⁷² The length of the catheter-over-needle devices makes them unsuitable for central-venous access. The insertion of catheter-over-guidewire, or Seldinger technique, is often the technique of choice for central-venous-catheter insertion.⁷³

Table 4. Advantages and Disadvantages of Femoral-Vein Cannulation

Advantages

- The femoral vein does not collapse like peripheral vessels.
- Easily identifiable landmarks allow proper cannulation.
- CPR is not interrupted during cannulation.
- The catheter may allow additional therapeutic interventions (ie, triple-lumen catheter).

Disadvantages

- In low-flow states, arterial pulse is decreased and femoral vein may pulsate leading to incorrect cannulation—also, arterial blood may look like venous blood (ie, dark color).
- Decreased extremity flow leads to prolonged time of drug entry into central compartment.
- A long catheter that extends above the diaphragm is required to ensure adequate central compartment entry.
- Femoral-vein cannulation poses greater risk of thrombosis and infection than peripheral sites.
- Phlebitis may extend to iliac veins and the inferior vena cava.

Volume Resuscitation

Any intravenous site provides a means of delivering fluids to the patient in shock. Estimates of fluid volume requirements are based on blood pressure, pulse, central-venous pressure, urine output, and neurologic function. The preferred solution in the cardiac-arrest setting is isotonic solutions—NS or Ringer's lactate.⁷⁴

Isotonic solutions initially distribute only into the extracellular fluid compartment. Water accounts for 60% of body weight—two thirds is intracellular fluid and one third is extracellular fluid. Of the extracellular fluid, 75% is interstitial and 25% is intravascular. Volume distribution is important to appreciate because hypotonic, or potentially hypotonic fluids, distribute into the total body water space. Hence, hypotonic (0.225% NS) or potentially hypotonic (dextrose in water, D₅W) solutions have only transient effects on intravascular volume. Less than 10% of the volume of D₅W that is administered remains in the intravascular space after infusion. Most of the intravenously administered isotonic crystalloid ultimately migrates to the interstitial space.

During resuscitation, NS is preferred unless the patient has hyperchloremic acidosis, in which case the salt load may worsen the problem.⁷⁵ In renal failure and hyperkalemic states, the potassium in Ringer's lactate may be dangerous. Although the lactate in Ringer's lactate does not potentiate systemic lactic acidosis, its metabolism to bicarbonate is slowed in patients who are in shock and those with liver hypoperfusion.

For vascular volume expansion, colloid (eg, albumin, dextran, hetastarch) is superior to crystalloid. Less than 25% of crystalloid remains in the intravascular space once

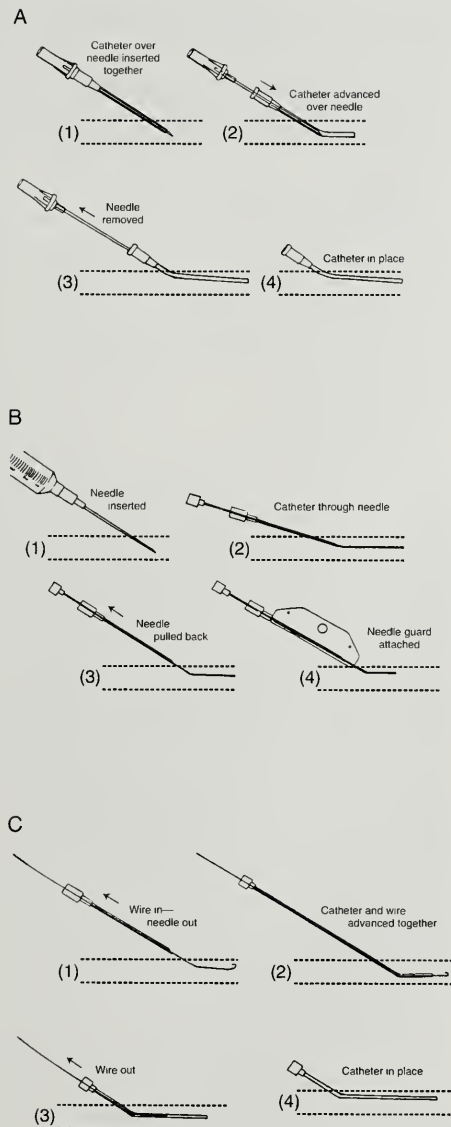


Fig 19. (A) Technique for insertion of catheter over needle. (B) Technique for insertion of catheter through needle. (C) Technique for insertion of catheter over guidewire. (Seldinger technique) (From Reference 61, with permission).

hour after administration, whereas infused colloid remains in the intravascular space for many hours. Colloids may produce volume expansion greater than the volume infused. In other words, 4-5 times more crystalloid than colloid is required to replace acute blood loss. However, colloid is expensive, poses an allergic risk, and is difficult to store and transport.⁷⁶

For hypotensive patients following penetrating trauma, it has been a common practice to provide prompt intravenous infusion of fluids to maintain organ perfusion and vital organ function.^{77,78} The concern with this practice is that it may disrupt the formation of thrombi, resulting in increased bleeding and decreased survival. A recently published prospective study of 598 hypotensive adults with penetrating torso injuries found that delay of aggressive fluid resuscitation until operative intervention resulted in an improved outcome.⁷⁸ However, further study is needed before changes in current recommendations for volume resuscitation are adopted.

Intracardiac Drug Administration

AHA Recommendations

The only drug administered by intracardiac injection during CPR is epinephrine. The AHA recommends that intracardiac injection only be used during open cardiac massage or when other routes of administration are unavailable.

Technique

Intracardiac injection is usually done by puncturing the left fourth intercostal space just lateral to the sternal border. The second through sixth intercostal spaces and sub-xiphoid approach have also been utilized. The choice among approaches depends on the administrator's experience and complication rate. Regardless of the mode of entry, the right-ventricular cavity is entered.⁷⁸ The left-ventricular cavity is penetrated usually only after penetration of the right ventricle or interventricular septum.⁷⁹ Brisk filling of the syringe indicates that the ventricular cavity has been entered and that therapy may be delivered. The close proximity of vital mediastinal and chest-wall structures may sometimes result in their inadvertent laceration. In addition, normal anatomical landmarks may be distorted by other cardiopulmonary and chest-wall disorders and may make rapid entry difficult.

The right ventricle is entered more often than the left ventricle because the former chamber occupies a greater portion of the anterior cardiac outline. Fluoroscopic guidance results in left ventricle entry approximately 50% of the time, but this is impractical during resuscitation. Injection into the left ventricle gives rapid delivery of a

drug to the coronary circulation without passing through the pulmonary vascular circuit. However, whether direct penetration of the left ventricle leads to any additional beneficial effect is not known.

Complications

Complications of intracardiac injection are numerous, but not as common as once thought.⁸⁰ Pneumothorax, hemopericardium, coronary artery laceration, and cardiac tamponade may result.^{81,82} Intramyocardial injection may precipitate cardiac dysrhythmias. The right ventricle is believed to have a decreased capacity to self-seal compared with the left ventricle. Although concern about hemopericardium remains, this notion has been challenged by a recent published report.⁸⁰

We presume that the increased intracoronary drug levels achieved by intracardiac administration may improve the outcome of resuscitation. Although with intracardiac injection, drugs reach the central circulation in a more predictable manner, use of this technique may increase myocardial ischemia and, in the cardiac-arrest setting, may lead to further compromise. In addition, intracardiac injection requires interruption of CPR during chest-wall entry, and such delays (> 5 s) may be deleterious.⁸³ The major disadvantage of intracardiac injection, according to Davison and colleagues,⁸⁰ is the need to interrupt CPR, not the risk of complications as was once believed.

Recommendations

Over recent years, central-vein cannulation has become safe and reliable for facilitating drug and fluid therapy and assessing the need for fluid therapy. Central veins serve as a route for pulmonary artery catheterization and temporary pacemaker electrode placement and provide an avenue to monitor acid-base status. The advantages of intracardiac injection have yet to be proven. There have been no clinical trials to compare intracardiac with intravenous therapy. Given the ease of administration of drugs via the central circulation or endotracheal tube and emerging knowledge about larger doses of epinephrine,⁸⁴ intracardiac injection cannot be recommended as routine practice and should remain as a procedure of last resort.

Role of the Respiratory Therapist

The respiratory therapist is an integral part of the resuscitation team. The specific responsibilities of the therapist vary, however, from hospital to hospital. It is commonly accepted that the respiratory therapist has primary responsibility for airway maintenance (including intubation in some hospitals) and ventilation (Table 5). The respiratory

therapist may also be responsible for drawing and analyzing arterial blood-gas samples.

Table 5. The Role of the Respiratory Therapist during In-Hospital Cardiopulmonary Resuscitation

Primary responsibilities

- Airway
- Ventilation
- Blood gases
- Instillation of endotracheal drugs

Secondary responsibilities

- Peripheral-vein catheter insertion
- Vascular drug infusion
- Defibrillation (may become a primary responsibility with increased use of automated defibrillators)

Unlikely responsibilities

- Central-Line insertion
- Intraosseous needle insertion
- Intracardiac injection

Because of their responsibilities related to airway management and ventilation, it is reasonable to expect that the respiratory therapist be responsible for emergency drug administration by endotracheal instillation. Drug administration via the airway route is already a responsibility of the respiratory therapist, who commonly administers aerosolized medications and may instill drugs, such as surfactant.

In some hospitals, respiratory therapists may have responsibilities such as insertion of peripheral-vein catheters and vascular-drug administration. However, this requires the presence of more than one therapist at the resuscitation (ie, one therapist to manage the airway and another to manage the intravenous line and drug administration). In reality, however, allowing the respiratory therapist to deal with airway management, ventilation, and blood gases and others to assume responsibility for intravenous-drug administration may be the most efficient use of personnel during resuscitations. As automated defibrillators become more commonplace, the respiratory therapist, as the first responder to the bedside of a patient in cardiac arrest, may assume responsibility for defibrillation.

Some nonphysician personnel (eg, paramedics) have been taught to insert central-vein catheters, insert intraosseous needles, and perform intracardiac injection, but it is unlikely that respiratory therapists will perform these tasks as a primary job responsibility. However, these are not complicated techniques and are within the scope of training and knowledge of many respiratory therapists.

REFERENCES

1. Zaritsky, A. Endotracheal epinephrine in cardiac arrest (editorial). *Crit Care Med* 1994;22:1071-1072.

2. Greenberg, MI. Endotracheal drugs: State of the art (editorial). *Ann Emerg Med* 1984;13:789-790.
3. Anonymous. Intratracheal drugs (editorial). *Lancet* 1988;1:743-744.
4. Redding JS, Asuncion JS, Pearson, JW. Effective routes of drug administration during cardiac arrest. *Anesth Analg* 1967;46:253-258.
5. Emergency Cardiac Care Committee and Subcommittees, American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiac care, III: Advanced cardiac life support. *JAMA* 1992;268:2199-2241.
6. Roberts JR, Greenberg MI, Knaub M, Baskin SI. Comparison of the pharmacological effects of epinephrine administered by the intravenous and endotracheal routes. *JACEP* 1978;7:260-264.
7. Roberts JR, Greenberg MI, Knaub MA, Zebulun KV, Baskin SI. Blood levels following intravenous and endotracheal epinephrine administration. *JACEP* 1979;8:53-56.
8. Greenberg MI, Roberts JR, Krusz JC, Baskin SI. Endotracheal Epinephrine in a canine anaphylactic shock model. *JACEP* 1979;8:500-503.
9. Greenberg MI, Roberts JR, Baskin SI. Use of endotracheally administered epinephrine in a pediatric patient. *Am J Dis Child* 1981;135:767-768.
10. Ralston SH, Voorhees WD, Babbs CF. Intrapulmonary epinephrine during prolonged cardiopulmonary resuscitation: Improved regional blood flow and resuscitation in dogs. *Ann Emerg Med* 1984;13:79-86.
11. Chernow B, Holbrook P, D'Angona DS, Zaritsky A, Casey LC, Fletcher JR, Lake CR. Epinephrine absorption after intratracheal administration. *Anesth Analg* 1984;63:829-832.
12. Ralston SH, Tacker WA, Carter A, Babbs CF. Endotracheal versus intravenous epinephrine during electromechanical dissociation with CPR in dogs. *Ann Emerg Med* 1985;14:1044-1048.
13. Hornchen U, Schuttler J, Stoeckel H, Eichelkraut W, Hahn N. Endobronchial instillation of epinephrine during cardiopulmonary resuscitation. *Crit Care Med* 1987;15:1037-1039.
14. Quinton DN, O'Byrne G, Aitkenhead AR. Comparison of endotracheal and peripheral intravenous adrenaline in cardiac arrest. *Lancet* 1987;1:828-829.
15. Crespo SG, Schoffstall JM, Fuhs LR, Spivey WH. Comparison of two doses of endotracheal epinephrine in a cardiac arrest model. *Ann Emerg Med* 1991;20:230-234.
16. Mazkereth R, Paret G, Ezra D, Aviner S, Peleg E, Rosenthal T, Barzilay Z. Epinephrine blood concentrations after peripheral bronchial versus endotracheal administration of epinephrine in dogs. *Crit Care Med* 1992;20:1582-1587.
17. McCririck A, Kestin I. Haemodynamic effects of tracheal compared with intravenous adrenaline. *Lancet* 1992;340:868-870.
18. Jasani MS, Nadkarni VM, Finkelstein MS, Mandell GA, Salzman S, Norman ME. Effects of different techniques of endotracheal epinephrine administration in pediatric porcine hypoxic-hypercarbic cardiopulmonary arrest. *Crit Care Med* 1994;22:1174-1180.
19. Brown LK, Diamond J. The efficacy of lidocaine in ventricular fibrillation due to coronary artery ligation: Endotracheal vs intravenous use. *Proc West Pharmacol Soc* 1982;25:43-45.
20. Melby MJ, Rachel CL, Kreul J. Pharmacokinetics of endotracheally administered lidocaine. *Clin Pharm* 1986;5:228-231.
21. Mace SE. Effect of technique of administration on plasma lidocaine levels. *Ann Emerg Med* 1986;15:552-556.
22. Hahnel JH, Lindner KH, Schurmann C, Pregel A, Ahnefeld FW. Endobronchial drug administration: does deep endobronchial delivery have advantages in comparison with simple injection through the endotracheal tube? *Resuscitation* 1990;20:193-202.
23. Hahnel JH, Lindner KH, Schurmann C, Pregel A, Ahnefeld FW. Plasma lidocaine levels and PaO_2 with endobronchial administration: dilution with normal saline or distilled water? *Ann Emerg Med* 1990;19:1314-1317.
24. Pregel AW, Lindner KH, Hahnel J, Ahnefeld FW. Endotracheal and endobronchial lidocaine administration: Effects on plasma lidocaine concentration and blood gases. *Crit Care Med* 1991;19:911-915.
25. Pregel AW, Lindner KH, Hahnel JH, Georgieff M. Pharmacokinetics and technique of endotracheal and deep endobronchial lidocaine administration. *Anesth Analg* 1993;77:985-989.
26. Greenberg MI, Mayeda DV, Chrzanowski R, Brumwell D, Baskin SI, Roberts JR. Endotracheal administration of atropine sulfate. *Ann Emerg Med* 1982;11:546-54.
27. Prete MR, Hannan CJ, Burkle FM. Plasma atropine concentrations via intravenous, endotracheal, and intraosseous administration. *Am J Emerg Med* 1987;5:101-104.
28. Rusli M, Spivey WH, Bonner H, McNamara RM, Aaron CK, Lathers CM. Endotracheal Diazepam: absorption and pulmonary pathologic effects. *Ann Emerg Med* 1987;16:314-318.
29. Barsan WG, Ward JT, Otten EJ. Blood levels of diazepam after endotracheal administration in dogs. *Ann Emerg Med* 1982;11:242-247.
30. Greenberg MI, Roberts JR, Baskin SI. Endotracheal naloxone reversal of morphine-induced respiratory depression in rabbits. *Ann Emerg Med* 1980;9:289-292.
31. Schanker L. Commentary. Drug absorption from the lung. *Biochem Pharm* 1978;27:381-385.
32. Bakkle YS. Pharmacokinetic and metabolic properties of lung. *Br J Anaes* 1990;65:79-93.
33. Greenberg MI, Spivey WH. Comparison of deep and shallow endotracheal administration of diazepam in dogs and effect of manual hyperventilation. *Ann Emerg Med* 1985;14:209-212.
34. Greenberg MI, Baskin SI, Kaplan AM, Urrichio FJ. Effects of endotracheally administered distilled water and normal saline on the arterial blood gases of dogs. *Ann Emerg Med* 1982;11:600-604.
35. Drinker CK, Drinker KR, Lund CC. The circulation in the mammalian bone marrow. *Am J Physiol* 1922;62:1-92.
36. Rosetti V, Thompson BM, Aprahamian C. Difficulty and delay in intravascular access in pediatric arrests (abstract). *Ann Emerg Med* 1984;13:406.
37. Tocantins LM, O'Neill JF, Jones HW. Infusions of blood and other fluids via the bone marrow. *JAMA* 1941;117:1229-1234.
38. Hodge D. Intraosseous infusions: A review. *Ped Emerg Care* 1985;1:215-218.
39. Rosetti VA, Thompson BM, Miller J, Mateer JR, Aprahamian. Intraosseous infusion: An alternative route of pediatric intravascular access. *Ann Emerg Med* 1985;14:885-888.
40. Spivey W. Intraosseous infusions. *J Pediatrics* 1987;111:639-643.
41. Fiser DH. Intraosseous infusion. *N Engl J Med* 1990;322:1579-1581.
42. Kruse JA, Vysokil JJ, Haupt MT. Intraosseous infusions: A flexible option for the adult or child with delayed, difficult, or impossible conventional vascular access. *Crit Care Med* 1994;22:728-729.
43. Neish SR, Macon MG, Moore JWM, Graeber GM. Intraosseous infusion of hypertonic glucose and dopamine. *AJDC* 1988;142:878-880.
44. Berg RA. Emergency infusion of catecholamines into bone marrow. *Am J Dis Child* 1984;138:810-811.
45. McNamara RM, Spivey WH, Unger HD, Malone DR. Emergency applications of intraosseous infusion. *J Emerg Med* 1987;5:97-101.
46. Orłowski JP, Porembka DT, Gallagher JM, Van Lente F. The bone marrow as a source of laboratory studies. *Ann Emerg Med* 1989;18:1348-1351.

47. Smith RJ, Keseg DP, Manley LK, Standeford T. Intraosseous infusions by prehospital personnel in critically ill pediatric patients. *Ann Emerg Med* 1988;491-495.
48. Seigler RS, Tecklenburg FW, Shealy R. Prehospital intraosseous infusion by emergency medical services personnel: A prospective study. *Pediatrics* 1989;84:173-177.
49. Miner WF, Corneli HM, Bolte RG, Lehnhof D, Clawson JJ. Prehospital use of intraosseous infusion by paramedics. *Ped Emerg Care* 1989;5:5-7.
50. Brickman K, Rega P, Choo M, Guinness M. Comparison of serum phenobarbital levels after single versus multiple attempts at intraosseous infusion. *Ann Emerg Med* 1990;19:31-33.
51. Valdes M. Intraosseous fluid administration in emergencies. *Lancet* 1977;1:1235-1236.
52. Iserson KV. Intraosseous infusions in adults. *J Emerg Med* 1989;7:587-591.
53. Shoor PM, Berryhill RE, Benumof JL. Intraosseous infusion: Pressure-flow relationship and pharmacokinetics. *J Trauma* 1979;19:772-774.
54. Hodge D, Delgado-Paredes C, Fleisher G. Intraosseous infusion flow rates in hypovolemic "pediatric" dogs. *Ann Emerg Med* 1987;16:305-307.
55. Schoffstall JM, Spivey WH, Davidheiser S, Lathers CM. Intraosseous Crystalloid and blood infusion in a swine model. *J Trauma* 1989;29:384-387.
56. Andropoulos DB, Soifer SJ, Schreiber MD. Plasma epinephrine concentrations after intraosseous and central venous injection during cardiopulmonary resuscitation in the lamb. *J Pediatrics* 1990;116:312-315.
57. Cameron JL, Fontanarosa PB, Passalacqua AM. A comparative study of peripheral to central circulation delivery times between intraosseous and intravenous injection using a radionuclide technique in normovolemic and hypovolemic canines. *J Emerg Med* 1989;7:123-127.
58. Warren DW, Kissoon N, Mattar A, Morrissey G, Gravelle D, Rieder M. Pharmacokinetics from multiple intraosseous and peripheral intravenous site injections in normovolemic and hypovolemic pigs. *Crit Care Med* 1994;22:838-843.
59. Spivey WH, Lathers CM, Malone DR, Unger HD, Bhat S, McNamara RN, Schoffstall J, Turner N. Comparison of intraosseous, central, and peripheral routes of sodium bicarbonate administration during CPR in pigs. *Ann Emerg Med* 1985;14:1135-1140.
60. Ballar D. Biopharmaceutical considerations in subcutaneous and intramuscular drug administration. *J Pharm Sci* 1968;57:357-378.
61. Textbook of Advanced Cardiac Life Support. American Heart Association, Dallas, 1994, 6-1 to 6-5.
62. Hershey CO, Tomford TW, McLaren CE, Porter DK, Cohen DI. The natural history of intravenous catheter-associated catheter-related phlebitis. *Arch Int Med* 1984;144:1373-1375.
63. Barson WG, Levey RC, Weir H. Lidocaine levels during CPR: Differences after peripheral venous, central venous and intracardiac injections. *Ann Emerg Med* 1981;10:73-78.
64. Emerman CL, Pinchak AC, Hancock D, Hagen JF. Effect of injection site on circulation times during cardiac arrest. *Crit Care Med* 1988;16:1138-1141.
65. Rosen M, Latto IP, Shang W. Percutaneous central venous cannulation. *Br Med J* 1980;281:372-375.
66. Mitchell SE, Clark RA. Complications of central venous catheterization. *Am J Roentgenol* 1979;133:467.
67. Jastermski MS, Matthias S, Randell PA. Femoral venous catheterization during cardiopulmonary resuscitation: a critical appraisal. *J Emerg Med* 1984;1:387-391.
68. Molinar PS, Belani KG, Buckley JJ. Delayed hydrothorax following percutaneous central venous cannulation. *Acta Anaesthesiol Scand* 1984;28:493-496.
69. Hentrich F, Laufkotter EA. Intravascular knotting of a subclavian catheter: nonsurgical removal (letter). *Chest* 1985;87:698.
70. Collier PE, Ryan JJ, Diamond DL. Cardiac tamponade from central venous catheters. Report of a case and review of the English literature. *Angiology* 1984;35:600.
71. Dodson T, Quindlen E, Cromwell R, McEnany MT. Vertebral arteriovenous fistulas following insertion of central monitoring catheters. *Surgery* 1980;87:343-346.
72. Dutky PA, Stevens SL, Maul KI. Factors affecting rapid fluid resuscitation with large-bore introducer catheters. *J Trauma* 1989;29:856-860.
73. Barker WJ. Central venous catheterization: internal jugular approach and alternatives. In: Roberts JR, Hedges, eds. *Clinical procedures in emergency medicine*. 2nd edition. WB Saunders, Philadelphia, 1991, 340-351.
74. Shine KI, Kuhn M, Young LS, Tillisch JH. Aspects of management of shock. *Ann Emerg Med* 1980;9:723-734.
75. Coran AG, Ballantine TV, Horwitz DL, Herman CM. The effect of crystalloid resuscitation in hemorrhagic shock on acid-base balance: a comparison between normal saline and Ringer's lactate solutions. *Surgery* 1971;69:874-880.
76. Virgilio RW, Rice CL. Crystalloid versus colloid resuscitation: is one better? A randomized clinical study. *Surgery* 1979;85:129-139.
77. Jacobs LM. Timing of fluid resuscitation in trauma (editorial). *N Engl J Med* 1994;331:1153-1154.
78. Bickell WH, Wall MJ, Pepe PE, Martin RR, Ginger VF, Allen MK, Mattox KL. Immediate versus delayed fluid resuscitation for hypotensive patients with penetrating torso injuries. *N Engl J Med* 1994;331:1105-1109.
79. Sabin HI, Khunt K, Coghill SB, McNeill GO. Accuracy of intracardiac injections determined by a post-mortem study. *Lancet* 1983;1054-1055.
80. Davison R, Barresi V, Parker M, Meyers SN, Talano JV. Intracardiac injections during cardiopulmonary resuscitation. A low risk procedure. *JAMA* 1980;244:1110-1111.
81. Frolich TG, Davison R. Intracardiac injections. In: Roberts JR, Hedges, eds. *Clinical procedures in emergency medicine*. 2nd edition. WB Saunders, Philadelphia, 1991, 377-382.
82. Pun KK. Cardiac tamponade after intracardiac injection. *Anaesth Intensive Care* 1984;12:66-67.
83. Kesszler H, Carroll GC. Intracardiac injections during resuscitation (letter). *JAMA* 1981;246:123.
84. Barton C, Callahan M. High-dose epinephrine improves the return of spontaneous circulation rates in human victims of cardiac arrest. *Ann Emerg Med* 1991;20:722-725.

Hess Discussion

Pepe: Dean, you obviously did a good job reviewing everything, and I just wanted to ask whether you found

any studies that demonstrated that intraosseous drug administration changed the outcome?

Hess: I did find a few anecdotal re-

ports, as was the case with intratracheal drug administration.

1. Valdes MM. Intraosseous fluid administration in emergencies. *Lancet* 1977;1:1235-1236.

2. McNamara RM, Spivey WH, Unger HD, Malone DR. Emergency applications of intraosseous infusion. *J Emerg Med* 1987; 5:97-101.

3. American Heart Association and The American Academy of Pediatrics. Textbook of pediatric advanced life support. Dallas and Elk Grove Village: 1988;44.

Pepe: Do you know of any that proved a change in outcome? I don't know of any controlled studies.

Hess: I don't either. I'm trying to think down through that literature. Some of it is very old and dates way back to the 1940s. It tends to consist primarily of descriptions of technique and is, by and large, anecdotal.

Pepe: Again, I don't know of any such work, and I'm only trying to emphasize that we need to study it.

Malinowski: I just wanted to make a comment on that. I think from my review of the pediatric literature, which is where intraosseous infusions are used most often, I found no controlled studies either, but there is the theoretical potential for intraosseous techniques to impact outcome, and I agree, we need to study it. It is difficult to establish vascular access in infants and children during resuscitation. One group reported that it took more than 10 minutes for the most experienced person to establish vascular access, and, in many cases, they were never successful.¹ In contrast to that, one of those studies, reported only in abstract form, showed that intraosseous infusions could be started within a minute or two.² Intraosseous access is recommended in the PALS standards.³ So, I think that outcome might be influenced by this technique.

Pepe: But again, faster access still doesn't answer the question as to whether it changes outcome. It does make it a better intervention to study, however.

Durbin: I'd like to make several comments. First, Joe Redding is from Baltimore City Hospital, not from Johns Hopkins, where he and Peter Safar did their pioneering work on mouth-to-mouth breathing. They recognized a good deal when they saw it. Since then, BCH has been purchased by Hopkins so, the history of CPR is still the *Baltimore* story, if you will, rather than the Hopkins story. Second, did you look at any respiratory therapy techniques for administering cardiac drugs, such as inhalation or nebulization? If getting the drug to the distal airway is your goal, rather than getting it into the trachea, do you think these techniques have any theoretical advantages?

Hess: I don't know that anybody has looked at that. I would think that the problem is that it would take too long to deliver the drug. The advantage of a bolus administration is you can squirt a bolus of drug into the lung quickly. With the typical nebulizer, less than 5% of the drug makes it through the endotracheal tube.¹ Also, it would take so long, I'm afraid, to deliver an adequate amount of epinephrine to the patient; I think it's unlikely to be of use. But I don't know that anybody has looked at that.

too conservative. Go for it! If a third-year medical student, paramedic, or physician can put in a central line and a situation arises in which a respiratory care practitioner is the senior person there, he or she should be allowed to do that, too. So I think an RCP should be taught and allowed to do anything that a paramedic, physician's assistant, medical student, critical care nurse, or even a doctor may do, given the proper training.

Hess: I agree with that. Certainly, respiratory therapists are familiar with arterial and venous access. We draw arterial blood gases. I've inserted arterial lines and I.V.s and so forth. The problem is, though, that in a cardiac arrest situation, if there's only one therapist present, I'd rather have the therapist doing what they're best at doing, which is ventilation and airway management, rather than have them turn that over to a less-skilled individual, so that they can start an I.V. or push a drug. Now, if it's a situation where the hospital can send more than one therapist to the code, that's different. But I can tell you that it's unlikely that there are going to be 2 or 3 therapists present so that they can manage the airway, start the I.V., and defibrillate the patient. I don't think there's any reason that therapists could not do those things, but I think the available manpower often makes it unlikely. Respiratory therapists can, and should, be taught ACLS skills. I think that therapists can perform any function at a cardiac arrest, provided they have been trained.

Kaye: Of course they can. Remember, this is the time to make broad recommendations, not to be too narrow. There may be only one therapist who may be the most qualified person to put in an I.V. The therapist may have to ask someone else, I've intubated; please ventilate this individual for a while, and let me put the I.V. in. Don't be too conservative.

1. Rosetti J, Thompson B, Aprahamian C. Difficulty and delay in intravascular access in pediatric arrest (abstract). *Ann Emerg Med* 1984;13:406.

2. Seigler R, Tecklenburg F, Shealy R. Prehospital Intraosseous infusion by emergency medical services personnel: a prospective study. *Pediatrics* 1989;84:172-177.

1. MacIntyre NR, Silver RM, Miller CW, Schuler F, Coleman RE. Aerosol delivery in intubated, mechanically ventilated patients. *Crit Care Med* 1985;13:81-84.

Kaye: I admire your list of procedures that you believe RCPs should be allowed to perform, but don't be

Bishop: Those of us who are in Seattle or who have been in Seattle know that anytime we're talking about the trachea, we have to invoke the bronchial circulation. I think one of the problems, as you pointed out, looking at studies not done during cardiac arrests versus studies done during arrests, is that uptake is probably via the bronchial circulation; and bronchial circulation undergoes changes under varying conditions. Studies during pulmonary artery occlusion demonstrate that the bronchial circulation drops off to almost nothing when pulmonary flow is occluded.¹ For all we know, the same thing may happen during an arrest, and there may be very little bronchial circulation, so I think it's a very different circumstance.

1. Kowalski TF, Guidotti S, Deffebach M, Bishop MJ. The bronchial circulation in pulmonary artery occlusion and reperfusion. *J Appl Physiol* 1990;68:125-129.

Hess: Your point's a good one. I think that drug uptake from the lung with intratracheal administration may be via the bronchial circulation and not through the pulmonary circulation at all.

Bishop: In terms of all the various methods of access, at least in adults,

femoral venous access is usually quite easy to secure because the femoral vein is often very large during arrest. Access can be gained fairly quickly, and that might be the site that we should be training people to go for fairly quickly.

Hess: That issue is addressed in the paper.

Kaye: Be careful about femoral venous access. During CPR blood flow below the diaphragms is poor. What you need to do is put in a long catheter that extends above the diaphragms. If you do angiography during CPR, blood regurgitates through the tricuspid valve into the inferior vena cava.^{1,2}

1. Niemann JT, Rosborough JP, Hausknecht M, Garner D, Criley JM. Pressure-synchronized cineangiography during experimental cardiopulmonary resuscitation. *Circulation* 1981;64(5):985-991.
2. Niemann JT, Rosborough JP, Ung S, Criley JM. Hemodynamic effects of continuous abdominal binding during cardiac arrest and resuscitation. *Am J Cardiol* 1984;53(2):269-274.

Hess: And again, this issue has been dealt with in the paper. The issue with femoral-vein access is, how far should the catheter extend? A long

catheter that extends above the diaphragm is required for rapid delivery of fluids and medications into the central circulation.

Boudin: I just wanted to make sure that the point you made about intratracheal administration of drugs was clear as it directly relates to the RCP's role. You mentioned that direct instillation of drugs via the endotracheal tube was better than via a catheter inserted into the endotracheal tube. However, regarding diluted vs undiluted drug, followed by a squirt of saline—the latter seems as though it would be the easier and less time-consuming method. Which is better: a drug-saline cocktail or a shot of drug followed by a saline chaser?

Hess: Yes, in the study by Mace!—One of the things that they studied was 3 mL instilled down the endotracheal tube followed with 3 mL normal saline. That was better than if they just squirted the drug down the endotracheal tube. But it was not as good as when they diluted the drug to 6 mL, and squirted that down the endotracheal tube as one bolus.

1. Mace SE. Effect of technique of administration on plasma lidocaine levels. *Ann Emerg Med* 1986;15:552-556.

Resuscitation: When is Enough, Enough?

Robin J Hamill MD

- I. Introduction
- II. Confounding Variables Affecting Resuscitation Success
 - A. Underlying Disease
 - B. Patient's Age
 - C. Patient's Right of Self-Determination
- III. Physiologic Variables Affecting Successful Resuscitation
 - A. Time to Initiation of CPR
 - B. Effectiveness of Ventilation
 - C. Effectiveness of Cardiac Massage
 - D. Duration of CPR
 - E. Medications Administered
 - F. Rhythm
 - G. Recurrent Arrests
 - H. Absence of Neurologic Function
- IV. In Summary

Introduction

Cardiopulmonary resuscitation (CPR), as we know it, was introduced by Jude, Kouwenhoven, and Knickerbocker in 1960 for victims of acute insults such as drowning, electrical shock, drug reaction, anesthetic mishap, heart block, and dysrhythmias associated with acute myocardial infarction. They addressed indications and contraindications in this way:¹

Not all dying patients should have cardiopulmonary resuscitation attempted. Some evaluation should be made before proceeding. The cardiac arrest should be sudden and unexpected. The patient should not be in the terminal stages of a malignant or other chronic disease, and there should be some possibility of a return to a functional existence. The rigid time limit of 3 to 5 minutes since the onset of arrest of cardiac

output should not be exceeded. In regard to the latter when there is a genuine question of the duration of arrest, resuscitation should be attempted.

Unfortunately, current practice often appears to fail to meet such criteria. With the widespread training of health care providers and the lay public and the development of code teams and arrest protocols, CPR has come to be used for virtually anyone found in a pulseless state, unless explicit documentation exists that the patient should not be resuscitated. Yet, survival rates have not improved dramatically in the past 30 years. Initial success with resuscitation ranges from 30 to 50%.²⁻⁵ Although survival to discharge has been reported to occur in as many as 29% of patients suffering cardiac arrest and undergoing cardiopulmonary resuscitation in the hospital,⁶ most studies report less encouraging results, with survival to discharge occurring in about 15% of resuscitated patients.^{2,4,7-9} Of the survivors, 10-20% have evidence of moderate-to-severe neurologic dysfunction.^{10,11} Thus, many patients undergo cardiopulmonary resuscitation that results in an expensive prolongation of life but with no impact on the ultimate outcome.

CPR is one of many therapeutic interventions available to the health-care provider for the management of patients. Like chemotherapy, CPR has specific indications, and situations exist in which utilization of the modality does not

Dr Hamill is Associate Professor, Anesthesiology and Critical Care Medicine, University of Virginia, Charlottesville, Virginia.

A version of this paper was presented by Dr Hamill during the RESPIRATORY CARE Journal Conference "Resuscitation in Acute Care Hospitals," held in Cancun, Mexico, October 21-23, 1994.

Reprints: Dr Robin J Hamill, Dept of Anesthesiology, University of Virginia Health Sciences Center, Box 238, Charlottesville VA 22908

alter outcome. As suggested by Brett,¹² no ethical imperative requires a physician to perform procedures in the absence of at least the likelihood of a "modicum of benefit." CPR can be a useful and sometimes lifesaving therapeutic tool that should be applied, judiciously and with forethought, to patients who have a reasonable chance of responding to the treatment.

Although acute return of spontaneous circulation (ROSC) is one measure of outcome resulting from effective cardiopulmonary resuscitation, for practical and economic reasons immediate success of the resuscitative effort is not the yardstick by which we should measure the effectiveness of CPR. Rather, the patient's survival to discharge in a neurologically functional state is a more germane assessment tool. In this paper, I attempt to identify those patients who are more likely to benefit from CPR in the event of a cardiopulmonary arrest within an acute care hospital and those physiologic and demographic variables that correlate with functional survival to discharge from the hospital. In addition, I discuss the variables that suggest the futility of active resuscitation efforts.

Confounding Variables Affecting Resuscitation Success

Underlying Disease

A number of studies have suggested that certain underlying conditions are associated with poor response to CPR (Table 1). These include the presence of end-stage disease (eg, congestive heart failure, hepatic failure), acute disease (eg, pulmonary embolism, acute myocardial infarction), neurologic disease (eg, cerebrovascular accident, muscular dystrophy), and acute deterioration (eg, progressive dyspnea). Patients with cancer are not likely to survive to dis-

Table 1. Diagnoses and Conditions associated with < 5% Survival to Discharge following Cardiac Arrest

Diagnosis/Condition	First Author
Cancer	Peatfield ² Bedell ⁸ Hershey ¹³
AIDS	Schwenzer ¹⁰
Dementia	Schwenzer ¹⁰
Pneumonia	Bedell ⁸
Sepsis	Taffet ⁴
Renal failure	Bedell ⁸
Gastrointestinal bleeding	Peatfield ²
Homebound, prehospital	Bedell ⁸
Age > 70 years	Taffet ⁴ Bonnin ¹³

charge.¹⁴ Castagna et al³ in their 1974 review of 1,063 arrests found that arrest on the hospital ward is associated with a survival of only 2.1%. Pneumonia has also been associated with poor response to resuscitative efforts.⁸ Although sepsis may be associated with a reasonable initial response (45% success), survival to discharge is unlikely (1.3%).⁴ Similarly, cardiac arrest in the face of gastrointestinal bleeding is associated with 0% survival to discharge.² The number of diagnoses (ie, comorbidities) also appears to affect response in that patients with more pathologic diagnoses are less likely to survive.¹⁰

A number of metabolic factors can influence immediate response to resuscitative efforts and, thus, influence outcome. Although hypothermic patients may be difficult to resuscitate because of resistant ventricular dysrhythmias, hyperkalemia with reperfusion, and hemodynamic instability, they have the benefit of a hypothermia-induced decrease in metabolic rate and, hence, oxygen consumption. Even a 2-3° C decrease in body temperature provides substantial protection against ischemia and hypoxemia.¹⁵ Oxygen consumption decreases 5-8% for every degree-centigrade decline in core temperature.¹⁶ On the other hand, patients with high metabolic rates become hypoxic more quickly and are more likely to have a significant deficiency of oxygen delivery even in the face of effective CPR. Fever, catecholamine release, and seizures all increase cerebral oxygen consumption and decrease the amount of time available before the protective cerebral penumbra is exceeded and neuronal integrity is jeopardized.¹⁷

Underlying disease influences clinical decisions regarding the administration and continuation of CPR (Table 2).¹⁰ Patients in arrest for whom no CPR intervention is provided often have diagnoses such as neoplastic disease, stroke, in-

Table 2. Selective Application of CPR by Disease Category and Associated Survival to Discharge*

Factor	Number	% CPR	% Discharged Alive
All Events†	1,756	31.3	25.1
MMP	614	33.9	23.1
Acute disease‡	342	43.9	27.3
Procedure-related	61	100.0	26.2
Congenital disease	136	30.9	50.0
Neoplasm	173	22.0	10.5
Neoplasm + metastases	234	6.4	20.0
Trauma	108	16.7	11.1
Burn	25	52.0	23.1
AIDS	22	13.6	0.0
Dementia	41	4.9	0.0

*Adapted from Reference 10, with permission.

†Event = patient who received in-hospital CPR or who died in the hospital without CPR. MMP = multiple medical problems.

‡Acute disease category does not include trauma patients.

tracranial hemorrhage, or dementia. When CPR is administered to patients with AIDS, dementia, or multiple trauma, the likelihood of survival to discharge is significantly less than it is for patients who arrest as a result acute disease, procedure-related complications, or anesthetic mishaps.¹⁰

Patient's Age

The effect of age on outcome from CPR after cardiac arrest is not clear. Some studies suggest that age is inversely correlated with survival to discharge.^{4,10,18} Taffet et al⁴ found that resuscitative efforts in patients aged 70 years or older resulted in a 0% survival rate compared to 16% in patients younger than 70 years of age. Conversely, Juchems et al¹⁹ found that survival for out-of-hospital arrests was not significantly affected by age but that survival to discharge after in-hospital arrest was significantly poorer in patients older than 70 years of age compared to those younger than 70 years (Fig. 1). Although not statistically significant, a trend is seen in some studies toward decreased survival in patients older than 70 years of age.²⁰ The Office of Technology Assessment, in its review of CPR in the elderly,²¹ has suggested

Elderly patients as a group have somewhat poorer outcomes than younger patients but the poorer outcomes in elderly patients reflect the higher prevalence of multiple diseases in these patients. Although the likelihood of multiple diseases increases with age, any particular older individual may not be affected.

Schwenzer and colleagues¹⁰ from my institution found that patients 70 years of age or older were less likely to be given CPR than those younger than 70 years (23.4% vs 35.3%, $p < 0.0001$), but that when CPR was administered, survival to discharge was not significantly less in the older group (21.9% vs 26.2%, $p = 0.34$). Of note, however, only 11.1% of patients 80 years of age or older received CPR, with a survival to discharge of 8.7%.¹⁰ In administering CPR to the elderly, one must keep in mind the high risk of rib and sternal fractures. A flailed chest or flailed sternum in an elderly patient with limited pulmonary reserve carries a high mortality. In one study,²² elderly patients who received mechanical ventilation for 3 or more days for respiratory failure had a 67% in-hospital mortality. Of the survivors, the 1-year survival was 16%, and the 3-year survival was only 9%.

Patient's Right of Self-Determination

Patients have the right of self-determination. In so far as is possible, the medical community should honor patient

preferences. It is unfortunate that all too often patients are not offered a choice as to how they are to be managed in the event of a cardiopulmonary arrest. Although advanced directives have made some inroads, in my experience, patients are rarely offered informed consent for CPR if and when they are asked about their wishes. Because CPR is an expensive intervention with limited success and a very real risk of survival with neurologic damage, patients should be allowed to make educated decisions. Thus, patients who have serious medical problems with poor prognosis and, particularly, those who are at risk of poor outcome from CPR should be offered the opportunity to refuse resuscitation should it become necessary (ie, 'informed consent'), at the earliest possible time after admission to the hospital.

Physiologic Variables Affecting Successful Resuscitation

Time to Initiation of CPR

The longer the interval between cardiac arrest and the initiation of effective CPR, the poorer the outcome (Fig. 2²³). Due to the widespread teaching of basic life support, it is not surprising that patients experiencing witnessed arrests have a better initial survival rate (47.7%) than those experiencing nonwitnessed arrests (29.9%, $p < 0.001$).⁴ Of the witnessed arrests in Taffet et al's study,⁴ 82% of those who survived initial resuscitation were discharged from the hospital alive ($p < 0.05$). Stults and colleagues²⁴ found that if the time from observed collapse to the time of first defibrillation was less

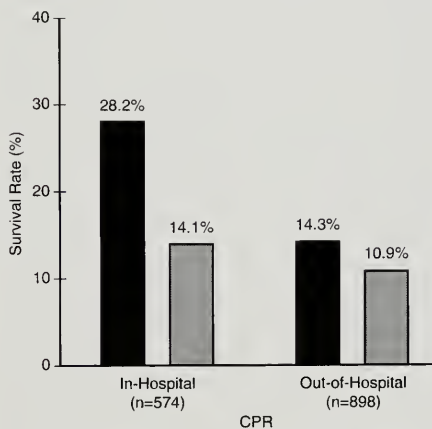


Fig.1. Survival rates by age in patients experiencing in-hospital and out-of-hospital arrest. ■ = < 70 years; □ = ≥ 70 years. (Adapted from Reference 19, with permission.)

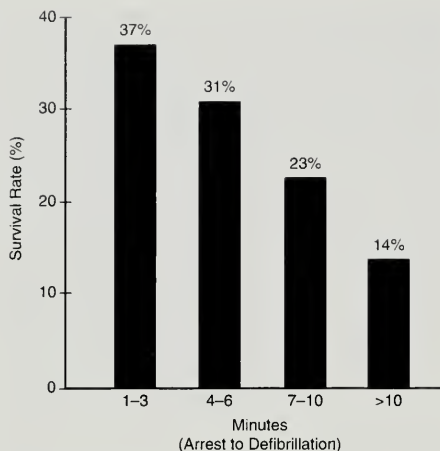


Fig. 2. Survival rate as a function of time from arrest to defibrillation: the greater the time, the lower the survival. (Adapted from Reference 23, with permission.)

than 6 minutes, then survival to discharge was 28%. On the other hand, if this interval was longer than 6 minutes, survival was less than 5%.²⁴ This suggests that, due to the extremely poor outcome in patients in whom the delay between arrest and defibrillation is prolonged *and well-documented* (particularly if bystander CPR was not administered), the rescuer should consider limiting the duration of the resuscitative effort unless the victim shows a rapid response to therapy.

In-hospital survival rates also depend on the location of the arrest. Hershey and Fisher¹⁴ found that survival to discharge occurred in only 3% of patients who arrested on the wards, whereas 16% of ICU patients and 21% of patients resuscitated in the emergency department survived to discharge. This is likely due to several factors including the increased likelihood of an observed arrest in the emergency department and intensive care unit and the higher level of monitoring employed in these locations. Therefore, the interval between arrest and initiation of resuscitative efforts is likely to be somewhat shorter in these settings compared to the wards. In addition, outcome is likely to be affected by the greater resuscitation experience of nursing personnel working in the acute care setting. Differences in diagnoses and etiologies of arrest may also contribute to these discrepancies. For example, patients who arrest in the midst of acute respiratory or metabolic deterioration have a 97% mortality.¹⁴

With cessation of blood flow, loss of consciousness occurs within 10 seconds—evidence of the brain's limited energy stores.²⁵ Electrical activity ceases when the cere-

bral blood flow decreases to less than $15 \text{ mL} \cdot \text{min}^{-1} \cdot 100 \text{ g}^{-1}$. As the blood flow diminishes further, cellular integrity is lost. Potassium leaks out of the cells, and calcium accumulates within them. The increased calcium uncouples oxidative phosphorylation and activates membrane phospholipases. Phospholipases dissolve cell membranes releasing fatty acids that are metabolized to leukotrienes, thromboxanes, and free radicals. With reperfusion, the increased calcium causes constriction of the precapillary sphincters. Cellular edema also forms in response to the accumulation of toxic metabolites and loss of membrane integrity. Neurologic outcome declines sharply if adequate perfusion is not established within 5 to 6 minutes of arrest. The neurologic complications following cardiac arrest include impaired memory, deficits in higher intellectual function, myoclonus, seizures, cortical blindness, spinal stroke, a persistent vegetative state, and brain death. Thus, if adequate perfusion is not established within the first 6 minutes, the rescuers should begin to re-evaluate the appropriateness of continuing the effort. Certainly, if there is good documentation that adequate perfusion has not been generated within 10 minutes, serious consideration should be given to discontinuation of resuscitation, particularly in patients who have other predisposing factors that increase their risk of neurologic deficits, such as advanced age and cerebrovascular disease that would limit flow even in the face of adequate CPR. Bonnin and colleagues¹³ found that all survivors of out-of-hospital arrests had ROSC within 25 minutes of arrest. Further, they found that, with the exception of ventricular fibrillation or tachycardia, failure to achieve ROSC for at least 5 minutes, at the scene, was consistent with a 100% mortality.

Effectiveness of Ventilation

The need for endotracheal intubation may be, in and of itself, of prognostic value. Bedell and co-workers⁸ found that patients who required intubation at the time of arrest had a 91% mortality compared to those who did not require intubation (mortality 51%). This suggests that patients who respond readily to resuscitative efforts and/or are readily oxygenated without invasion of the trachea have a better prognosis. These results are not likely to indicate that the procedure of securing the airway, in and of itself, is a lethal intervention. However, detection of esophageal placement of the endotracheal tube can be difficult even by experienced personnel and in the controlled setting of the operating suite.²⁶ The arrest situation is further complicated by higher levels of ambient noise and activity, diminished breath sounds, and excessive rhonchi due to aspiration or secretions that may make auscultatory evaluation of tube placement nearly impossible. Unrecognized esophageal intubation has disastrous implications and may account for a

small number of failed resuscitations but is not likely to account for the high mortality found by Bedell et al.⁸

Many of the clinical indicators of appropriate endotracheal intubation may be absent or difficult to interpret during CPR. Capnography can be a useful confirmatory tool in the controlled setting of the operating room or intensive care unit. During the arrest situation, however, its usefulness may be limited.

As noted earlier, many of the clinical indicators of appropriate endotracheal tube position may be absent or difficult to interpret during CPR. Capnography can be useful in recognizing esophageal intubation. Air in the esophagus has a very low CO_2 concentration. The amount of CO_2 expired depends on a number of factors including metabolic status, cardiovascular function, and ventilation. As metabolic rate increases, at a fixed minute ventilation, end-tidal P_{CO_2} (Pet_{CO_2}) increases. This can be seen, for example, with fever, malignant neuroleptic syndrome, and malignant hyperthermia. If cardiovascular function is inadequate such that pulmonary perfusion is impaired, then less CO_2 is delivered to the lung vasculature to be excreted into the alveoli and subsequently expired. Patients in shock or with pulmonary embolism manifest Pet_{CO_2} measurements that can be substantially less than arterial values. Similarly, if effective cardiac function ceases, as in the case of a cardiac arrest, exhaled CO_2 decreases dramatically.²⁷ Exhaled CO_2 may also be falsely low compared to the CO_2 in the blood of patients with increased shunts as the pulmonary blood flow bypasses the perialveolar capillaries and fails to diffuse into the alveoli.

Effectiveness of Cardiac Massage

The perfusion pressure generated by standard chest compression is usually less than 40% of normal values. A coronary perfusion gradient (diastolic aortic pressure minus right atrial pressure) of 15 to 18 mm Hg or more is usually associated with successful resuscitation.²⁸⁻³⁰ Thus, cardiac massage technique can significantly affect outcome. Compression applied over the lower one third of the sternum provides higher systolic pressures than more cephalad compression. The depth of compression, the force applied, and the rate of compression also impact on perfusion pressures.^{31,32} Thus, rescuers should attempt to use higher rates when performing CPR because the higher perfusion pressures achieved are more likely to lead to more adequate coronary pressures and a higher likelihood of ROSC.

Although chest compression can provide an arterial systolic pressure that is within the normal range, the diastolic pressure is usually much lower than normal. Palpation of a carotid artery pulse, the previous measure of effective CPR, correlates poorly with coronary or cerebral

perfusion pressures due to the lower mean pressure imposed by a depressed diastolic pressure. However, it has been suggested that the massive sympathetic stimulation associated with cardiac arrest and the administration of sympathomimetic drugs causes vasoconstriction of many nonessential vascular beds and may give preferential perfusion to the coronary and cerebral vasculature.^{33,34}

A number of modifications have been applied to the resuscitative protocol in an attempt to improve cardiac output and, consequently, coronary and cerebral perfusion. High-impulse CPR (rate = 120 compressions/min) causes a significant increase in systolic blood pressure (80 ± 39 mm Hg) compared to 61 ± 29 mm Hg for conventional CPR, ($p < 0.01$) and diastolic pressure (19 ± 13 mm Hg vs 13 ± 10 mm Hg, respectively, $p < 0.005$). Similarly, coronary perfusion gradient increases from 9 ± 11 mm Hg to 14 ± 15 mm Hg ($p < 0.005$) when the rate of compressions is increased from 60 to 120/min.³¹ Acute volume loading has also been used in an attempt to improve perfusion pressures during resuscitation. In the hypovolemic or hemorrhaging patient this is clinically indicated. The effects on euvolemic patients, on the other hand, are not well studied. In one study of induced ventricular fibrillation in otherwise healthy swine, Jameson and colleagues³⁵ found that early volume expansion with hypertonic saline and dextran did not improve coronary perfusion pressure, rate of ROSC or rate of short-term survival.

Simultaneous inflation of the lungs and compression of the chest has shown conflicting results. Rudikoff et al³⁶ found that this technique causes a doubling of aortic systolic pressure and a threefold increase in carotid blood flow. Similar results are found when the intrathoracic pressure is increased through tight abdominal binding to limit paradoxical movement of the diaphragm. Here the carotid blood flow doubled. However, Martin et al³⁷ reported that simultaneous ventilation and compression worsened coronary perfusion gradient with lethal results in all patients in this small study. Other studies have also shown a higher mortality rate in the simultaneous ventilation and compression group compared to standard CPR.³⁸ Application of CPR via pneumatic vest generates pressures similar to conventional CPR. Simultaneous ventilation with the vest causes a significant increase in aortic systolic pressure but causes a dramatic decrease in coronary perfusion gradients.³¹

Interposed abdominal compressions (a method of manual abdominal compression administered during the release phase of chest compression, added to conventional CPR) have been shown to improve multiple hemodynamic parameters compared to conventional CPR alone. These include systolic pressure (35%), diastolic pressure (100%), cardiac output (100%), and oxygen delivery (50%).³⁹ Pet_{CO_2} has also been used to compare the efficacy of these techniques. Patients receiving interposed compressions

had values that were 78% higher than those achieved during conventional CPR (17.1 torr vs 9.6 torr). In addition, the return of spontaneous circulation occurred 5 times more often in the combination group.⁴⁰

A number of investigators have studied the use of capnography as a measure of the adequacy of chest compressions and survival prediction. When chest compressions are inadequate in depth or frequency, PetCO_2 decreases. As the rescuer providing cardiac massage fatigues, it has been shown that PetCO_2 diminishes.⁴¹ On the other hand, a rapid rise in PetCO_2 is seen when an effective cardiac rhythm is re-established. This is often associated with an 'overshoot' phenomenon that returns to baseline within 5 minutes.²⁷ Patients who were unsuccessfully resuscitated maintained a plateau PetCO_2 value of $0.7 \pm 0.4\%$ whereas those who were resuscitated had values of $1.3 \pm 0.5\%$ during CPR and stabilized at $2.4 \pm 1.8\%$ within minutes of the return of a spontaneous rhythm. Other studies have confirmed that a PetCO_2 of 2% or more is associated with successful resuscitation in at least 50% of patients. Values less than 2% have nearly a 100% mortality.⁴² Interpretation of PetCO_2 may be difficult in the clinical setting, however. As noted earlier, esophageal intubation may provide (at least temporarily) a misleadingly low value. Administration of NaHCO_3 causes an acute increase, and alpha adrenergic agents a decrease in PetCO_2 values.⁴³ Based on this information, PetCO_2 cannot be used as the sole criterion for stopping CPR.

Other techniques including use of an active compression-decompression device^{44,45} and portable extracorporeal pump oxygenation (ECPO)^{46,47} are in the early stages of evaluation. Whereas active compression-decompression appears to offer significant promise of augmenting hemodynamic response to CPR and facilitating successful resuscitation, ECPO has been studied less and with conflicting results. Additional clinical experience is needed to determine the short- and long-term implications of these newer modalities.

Duration of CPR

Duration of CPR also impacts on survival. Inadequate myocardial perfusion causes a rapid decrease in myocardial compliance, thereby limiting cardiac filling and subsequent forward flow. In addition, progressive chest deformation and, in particular, a flailed chest and/or flailed sternum disrupt the elastic recoil of the thoracic wall. This, in turn, decreases venous return. In addition, with progressive acidemia, venous capacitance increases, further retarding venous return. Bedell and co-workers⁸ report that patients who received CPR for longer than 30 minutes without the return of spontaneous cardiac function were not resuscitated. 95% of those patients who required resuscitation for more than 15 minutes died. Conversely, only 44% of those

who developed a normal cardiac rhythm and effective perfusion within 15 minutes died. Of patients with active pre-morbid lifestyles who were resuscitated within 15 minutes, mortality was only 17%. Taffet et al⁹ compared the duration of CPR in survivors to nonsurvivors (Table 3).

Table 3: Outcome from CPR: Duration and Number of Medication Doses*

	Live Discharge	Death	p value
Duration of CPR (minutes)	13.6	22.4	<0.01
Doses of medication (number)	3.33	7.93	<0.01

*Adapted from Reference 4, with permission.

Medications Administered

One of the primary determinants of successful CPR is the rapid establishment of adequate coronary perfusion pressures to maintain myocardial viability. Drugs that increase mean arterial blood pressure also increase coronary perfusion (Fig. 3³⁴). Hence, use of alpha adrenergic medications such as epinephrine, norepinephrine, phenylephrine, or methoxamine (a pure alpha-1 agonist) has been advocated.⁴⁸ Although epinephrine is routinely used, the ideal dose has been debated. Animal studies show greater increases in cerebral blood flow in swine receiving high-dose epinephrine (0.2 mg/kg) compared to those receiving low-dose epinephrine (0.02 mg/kg) or methoxamine (10

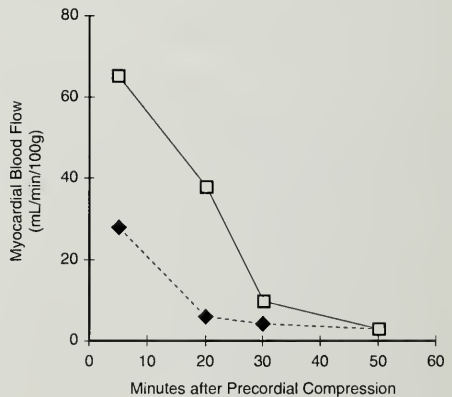


Figure 3. Effect of epinephrine on myocardial perfusion. Cardiac massage was initiated at Time 0, 10 minutes after arrest. Dogs who received epinephrine □ showed markedly higher myocardial perfusion up to 20 minutes after initiation of resuscitation than did dogs not receiving epinephrine ◆ ($p < 0.005$) (Adapted from Reference 34, with permission.)

mg/kg).⁴⁹ However, a subsequent study by Berg and colleagues,⁵⁰ found equally successful resuscitation with low- and high-dose epinephrine in swine. Of note, the high-dose animals had greater problems with severe hypertension (diastolic pressure > 120 mm Hg) and tachycardia (heart rate > 250 beats/min) as well as a higher early mortality compared to the low-dose group (28% vs 0%). In humans who experienced prehospital arrest, high-dose epinephrine (15 mg) and norepinephrine (11 mg) were compared to standard-dose epinephrine (1 mg). Almost twice as many patients receiving high-dose epinephrine regained a pulse than did the low-dose epinephrine group. The norepinephrine patients tended to be more likely to have return of a palpable pulse than the low-dose epinephrine patients, but less than the high-dose epinephrine group. This probably reflects the increased oxygen extraction associated with the administration of norepinephrine, which counteracts the effect of the coincident increased oxygen delivery.⁵¹ Unfortunately, no significant difference was seen between the groups with regard to survival to discharge from the hospital. Survival rates in all groups were low (1.2-2.6%).⁵²

Although calcium chloride has been used for the treatment of asystole and electromechanical dissociation (EMD), results do not conclusively show a positive effect on outcome. High levels of ionized calcium (4.3 mmol/L) can cause cerebral vasospasm.⁵³ In addition, high extracellular calcium concentrations can promote myocardial cell damage upon reperfusion.⁵⁴

The number of doses of medication administered also has prognostic significance (Table 3). Fewer doses prior to ROSC suggest the presence of a cardiac rhythm that responds more readily to treatment or an arrest due to problems that are easily corrected. Although there is a trend toward worse survival rates with higher numbers of doses,⁴ it is impossible to set a threshold for the number of doses of medication that would equate with a 0% survival.

Rhythm

Initial documented rhythms at the time of cardiac arrest include ventricular fibrillation, asystole, idioventricular rhythm, EMD, and ventricular tachycardia (Table 4³⁵). Response to resuscitation depends in part on the nature of the dysrhythmia. Eisenberg and colleagues⁵⁶ found better outcomes when patients were found to have ventricular tachycardia or ventricular fibrillation rather than asystole, EMD, or idioventricular rhythm. This may be due to the availability of better treatment options for the former rhythm disturbances or the presence of more ominous disease for the latter. The acute diagnosis of the etiology of rhythms such as EMD may be difficult. Further, sufficiently rapid treatment of massive pulmonary embolism, profound anemia, and/or hypovolemia, for example, may not

be accomplished within the limited time frame imposed by finite neurologic or myocardial energy stores.

Table 4. Incidence and Outcome according to Initial Dysrhythmias for Out-of-Hospital Arrests*

Rhythm	n	Incidence (%)	Survival to Discharge (%)
Ventricular fibrillation	1,238	58	31
Ventricular tachycardia	50	2	46
Asystole	576	27	2
Idioventricular or electromechanical dissociation	162	8	3
Other/Unknown	125	6	15
Total	2,151	—	21

*Adapted from Reference 55, with permission.

Recurrent Arrests

Repeated arrests are generally ominous, suggesting failure and/or inability to correct the underlying precipitating pathology. Bonnin and colleagues¹³ found that ROSC (for 5 minutes) within 25 minutes of arrest was a prerequisite for survival in their subset of patients with refractory ventricular fibrillation who survived. It was not clear from their paper whether there were intermittent periods of ROSC with degeneration to ventricular fibrillation or persistent ventricular fibrillation. If there is ROSC with degeneration to a nonperfusing rhythm does this constitute a repeat arrest? How long does the interval between arrests need to be to qualify them as separate events? These data do not exist. It has been suggested that patients suffering second and third arrests do not survive to leave the hospital (Table 5).³ There has been insufficient research, largely due to small numbers of patients who undergo multiple arrests, to be able to draw firm conclusions with regard to the appropriateness of CPR under these circumstances. Each case must be evaluated individually with regard to the potential for correction of the underlying pathology.

Table 5. Survival after Repeated Cardiopulmonary Resuscitation*

	Number of Resuscitations			
	1	2	3	Total
Attempted resuscitations	137	10	3	150
Success each attempt (%)	35	40	33	35
Long-term survivors (number)	14	0	0	14

*Adapted from Reference 3, with permission.

Absence of Neurologic Function

Evaluation of neurologic function may be particularly difficult during an arrest or immediately after successful resuscitation. Sympathomimetic drugs such as epinephrine may limit evaluation of pupillary response to light, and depression of mental status in the peri-arrest period is common. One study that evaluated patients upon arrival at the hospital after prehospital arrest found that the only early predictor of poor outcome was the absence of spontaneous respiration.¹⁸ The presence of consciousness upon admission was associated with a 4% mortality prior to discharge whereas those with an altered state of consciousness had a 53% in-hospital mortality. Failure to respond to painful stimuli and absence of the pupillary light reflex were not predictive of poor outcome until 48 hours after the arrest. The likelihood of permanent neurologic deficits increases with the duration of coma. Only 5% of patients who are unconscious at 48 hours will have full neurologic recovery.³⁷ Other authors have found that, by the third day following arrest, lack of motor response to pain, absence of pupillary light reflex, or the presence of a Glasgow Coma Scale score of < 6 were followed by a persistent vegetative state in all patients.⁵⁸ Posthypoxia myoclonus, once thought to be indicative of a grim prognosis, has been reported to resolve in a few patients with return of normal intellectual function but with a moderate residual movement disorder.⁵⁹

In Summary

Almost half of patients respond acutely to resuscitation but most die within the first several days after arrest. The incidence of survival to discharge from the hospital after cardiopulmonary arrest is about 15%; one third of those surviving have evidence of neurologic deficits. Although some prognostic variables are useful in determining which patients are most likely to die prior to discharge from the hospital, each patient needs to be evaluated on an individual basis and the various risk factors weighed carefully. As additional data accumulate, we may well be more effective at deciding which patients are more likely to benefit from CPR so that we can more judiciously apply this therapeutic modality.

A number of studies have identified factors that contribute to poor outcome. Patients over 70 years of age usually fare poorly after CPR, but this is more a reflection of the number of coexisting diagnoses rather than years. Although initial survival may not be different from younger patients, fewer elderly patients live to discharge and more are likely to have neurologic sequelae. Concurrent diagnoses such as sepsis, AIDS, gastrointestinal bleeding, renal failure, cancer, and central nervous system disease have a universally poor response to CPR. If

defibrillation occurs more than 6 minutes after arrest or on the general ward or if the resuscitative attempt lasts longer than 15 minutes, mortality is greater than 95%. If CPR continues for more than 30 minutes, there are no survivors.

A low exhaled CO₂ concentration (< 2%) during cardiac massage, asystole or EMD as the first identified rhythm, and recurrent arrest also carry a poor prognosis. On the other hand, at the time of arrest or during the immediate postarrest period, poor neurologic status is a less helpful predictor. The absence of spontaneous respiration is the only variable at the time of admission after out-of-hospital arrest that is particularly ominous. There is no evidence to suggest that the absence of spontaneous respiration implies any better prognosis for patients arresting in the hospital. Coma, hypoxic myoclonus, and absent reflexes, while not useful immediately following arrest, are of greater prognostic significance 48 hours later. Only 5% of patients who are unconscious 48 hours after arrest will have a full neurologic recovery. The Glasgow Coma Scale has also been used for prognostication. By the third day after arrest, a Glasgow score < 6 is followed, at best, by a persistent vegetative state.

Finally, more selective application of CPR is probably the best way to improve outcome. When patients suffer from end-stage disease, malignancy, acquired immunodeficiency syndrome, or other problems that are associated with poor response to CPR, they should be given the opportunity to determine their own code status. Where possible, it is the obligation of the physician to educate patients with regard to the reality of CPR—the process, and the aftermath including broken bones, tubes, neurologic damage and likelihood of death (or vegetative survival). This is particularly important for patients at high risk for poor outcome. CPR should be offered in situations in which it has potential benefit, like any other therapeutic modality. Initiation of futile or near-futile CPR because of family demands or because the physician has failed to address the issue with the patient and/or family prior to the event should occur less frequently.

On the other hand, outcome is improved by early defibrillation. High-impulse CPR with interposed abdominal compression leads to higher myocardial and cerebral perfusion pressures, the implication being a higher likelihood of resuscitation. It has been shown that a coronary perfusion-pressure gradient greater than 15 mm Hg is likely to be associated with successful resuscitation. Other techniques including the active compression-decompression device and portable extracorporeal pump oxygenation have not been adequately studied to determine the exact effect on outcome. However, the former looks much more promising than the latter. Perhaps, as technology evolves, we will have modalities that can impact mortality from cardiac arrest in a meaningful way.

Continuing improvement in prediction of outcome and assessment of benefits of new techniques will allow more informed choices by patients and caregivers in providing or not providing this costly therapy. Earlier prediction—during actual resuscitation—of ultimate futility is needed to help decide when enough is enough.

REFERENCES

- Jude J, Kouwenhoven W, Knickerbocker G. Cardiac arrest: report of application of external cardiac massage on 118 patients. *JAMA* 1961;178:1063-1070.
- Peatfield RC, Sillet RW, Taylor D, McNichol MW. Survival after cardiac arrest in hospital. *Lancet* 1977;1(8024):1223-1225.
- Castagna J, Weil MH, Shubin H. Factors determining survival in patients with cardiac arrest. *Chest* 1974;65(5):527-529.
- Taffet GE, Teasdale TA, Luchi RJ. In-hospital cardiopulmonary resuscitation. *JAMA* 1988;260(14):2069-2072.
- Berger R, Kelley M. Survival after in-hospital arrest in noncritically ill patients. A prospective study. *Chest* 1994;106:872-879.
- Robinson G, Hess D. Postdischarge survival and functional status following in-hospital cardiopulmonary resuscitation. *Chest* 1994;105:991-996.
- Schein RM, Hazday N, Pena M, Ruben BH, Sprung CL. Clinical antecedents to in-hospital cardiopulmonary arrest. *Chest* 1990;98(6):1388-1392.
- Bedell SE, Delbanco TL, Cook EF, Epstein FH. Survival after cardiopulmonary resuscitation in the hospital. *N Engl J Med* 1983;309(10):569-576.
- Ballew K, Philbrick J, Schorling J. Predictors of survival following in-hospital cardiopulmonary resuscitation. *Arch Intern Med* 1994;154:426-432.
- Schwenzer KJ, Smith WT, Durbin CG Jr. Selective application of cardiopulmonary resuscitation improves survival rates. *Anesth Analg* 1993;77(3):478-484.
- Messert B, Quaglieri C. Cardiopulmonary resuscitation: perspectives and problems. *Lancet* 1976;ii:410-412.
- Brett AS, McCullough LB. When patients request specific interventions: defining the limits of the physician's obligation. *N Engl J Med* 1986;315(21):1347-1351.
- Bonnin M, Pepe P, Kimball K, Clark P. Distinct criteria for termination of resuscitation in the out of hospital setting. *JAMA* 1993;270:1457-1462.
- Hershey CO, Fisher L. Why outcome of cardiopulmonary resuscitation in general wards is poor. *Lancet* 1982;1(8262):31-34.
- Minamisawa H, Smith M, Siesjo B. The effect of mild hypothermia and hypothermia on brain damage following 5, 10, and 15 minutes of forebrain ischemia. *Ann Neurol* 1990;28:26.
- Sessler D. Temperature monitoring. In: *Anesthesia*, R Miller, ed. New York: Churchill Livingstone 1994:1370.
- Gilston A. The eyes, the brain, and cardiac resuscitation. *Resuscitation* 1994;27:77-79.
- Bertini G, Margheri M, Giglioli C, Cricelli F, DeSimone L, Taddei T, et al. Prognostic significance of early clinical manifestations in postanoxic coma: a retrospective study of 58 patients resuscitated after prehospital cardiac arrest. *Crit Care Med* 1989;17(7):627-633.
- Juchems R, Wahlig G, Frese W. Influence of age on the survival rate of out-of-hospital and in-hospital resuscitation. *Resuscitation* 1993;26(1):23-29.
- Wackman R, Socaris S, Cohen I, Fein I. Survival from in-hospital cardiopulmonary resuscitation (CPR) in the geriatric population (abstract). *Crit Care Med* 1989;17:S77.
- Life-Sustaining Technologies and the Elderly, publication OTA-BA-306. US Congress, Office of Technology Assessment, 1987:167-201.
- Elpern EH, Larson R, Douglass P, Rosen RL, Bone RC. Long-term outcomes for elderly survivors of prolonged ventilator assistance. *Chest* 1989;96(5):1120-1124.
- Weaver W, Cobb L, Hallstrom A, Fahrenbruch C, Copassmk, Ray R. Factors influencing survival after out-of-hospital cardiac arrest. *J Am Coll Cardiol* 1986;7(4):752-757.
- Stults KR, Brown DD, Schug VL, Bean JA. Prehospital defibrillation performed by emergency medical technicians in rural communities. *N Engl J Med* 1984;310(4):219-223.
- Bass E. Cardiopulmonary arrest: pathophysiology and neurologic complications. *Ann Intern Med* 1985;103(6, Part 1):920-927.
- Keenan RL, Boyan CP. Cardiac arrest due to anesthesia: a study of incidence and causes. *JAMA* 1985;253(16):2373-2377.
- Falk JL, Rackow EC, Weil MH. End-tidal carbon dioxide concentration during cardiopulmonary resuscitation. *N Engl J Med* 1988;318(10):607-611.
- Ralston SH, Voorhees WD, Babbs CF. Intrapulmonary epinephrine during prolonged cardiopulmonary resuscitation: improved regional blood flow and resuscitation in dogs. *Ann Emerg Med* 1984;13(2):79-86.
- Niemann JT, Rosborough JP, Criley JM. Coronary perfusion pressure during cardiopulmonary resuscitation: a determinant of successful defibrillation after prolonged arrest and CPR (abstract). *Clin Res* 1984;32:208A.
- Paradis NA, Martin GG, Rivers EP, Goetting MG, Appleton TJ, Feingold M, Nowak RM. Coronary perfusion pressure and the return of spontaneous circulation in human cardiopulmonary resuscitation. *JAMA* 1990;263(8):1106-1113.
- Swenson RD, Weaver WD, Niskanen RA, Martin J, Dahlberg S. Hemodynamics in humans during conventional and experimental methods of cardiopulmonary resuscitation. *Circulation* 1988;78(3):630-639.
- Babbs CF, Voorhees WD, Fitzgerald KR, Holmes HR, Geddes LA. Relationship of blood pressure and flow during CPR to chest compression amplitude: evidence for an effective compression threshold. *Ann Emerg Med* 1983;12(9):527-532.
- Voorhees WD, Babbs CF, Tacker WA Jr. Regional blood flow during cardiopulmonary resuscitation in dogs. *Crit Care Med* 1980;8(3):134-136.
- Schleien CL, Dean JM, Koehler RC, Michael JR, Chantarojanasiri T, Traystman R, Rogers MC. Effect of epinephrine on cerebral and myocardial perfusion in an infant animal preparation of cardiopulmonary resuscitation. *Circulation* 1986;73(4):809-817.
- Jameson S, Mater J, DeBehnke D. Early volume expansion during cardiopulmonary resuscitation. *Resuscitation* 1993;26:243-250.
- Rudikoff MT, Maughan WL, Efron M, Freund P, Weisfeldt ML. Mechanisms of blood flow during cardiopulmonary resuscitation. *Circulation* 1980;61(2):345-352.
- Martin GB, Carden DL, Nowak RM, Lewinter JR, Johnston W, Tomlanovich MC. Aortic and right atrial pressures during standard and simultaneous compression and ventilation CPR in human beings. *Ann Emerg Med* 1986;15(2):125-130.
- Krischer JP, Fine EG, Weisfeldt ML, Guerci AD, Nagel E, Chandra N. Comparison of prehospital conventional and simultaneous compression-ventilation cardiopulmonary resuscitation. *Crit Care Med* 1989;17(12):1263-1269.

39. Voorhees WD, Niebauer MJ, Babbs CF. Improved oxygen delivery during cardiopulmonary resuscitation with interposed abdominal compressions. *Ann Emerg Med* 1983;12(3):128-135.
40. Ward KR, Sullivan RJ, Zelenak RR, Summer WR. A comparison of interposed abdominal compression CPR and standard CPR by monitoring end-tidal PCO₂. *Ann Emerg Med* 1989;18(8):831-837.
41. Kalenda Z. The capnogram as a guide to the efficacy of cardiac massage. *Resuscitation* 1978;6(4):259-263.
42. Varon AJ, Morrina J, Civetta JM. Clinical utility of a colorimetric end-tidal CO₂ detector in cardiopulmonary resuscitation and emergency intubation. *J Clin Monit* 1991;7(4):289-293.
43. Trillo G, von Planta M, Kette F. ETCO₂ monitoring during low flow states: clinical aims and limits. *Resuscitation* 1994;27(1):1-8.
44. Pell AC, Pringle SD, Guly UM, Steedman DJ, Robertson CE. Assessment of the active compression-decompression device (ACD) in cardiopulmonary resuscitation using transoesophageal echocardiography. *Resuscitation* 1994;27(2):137-140.
45. Cohen TJ, Tucker KJ, Lurie KG, Redberg RF, Dutton JP, Dwyer KA, and the Cardiopulmonary Resuscitation Working Group. Active compression-decompression: a new method of cardiopulmonary resuscitation. *JAMA* 1992;267(21):2916-2923.
46. Gazmuri R, Weil M, Gazmuri R, et al. End-tidal PCO₂ for monitoring cardiac resuscitation during extracorporeal pump oxygenation (abstract). *Clin Res* 1988;36:863A.
47. Hartz R, LoCicero J, Sanders J, et al. Portable bypass does not improve survival in cardiac arrest patients (abstract). *J Am Coll Cardiol* 1989;13:121A.
48. Raeli CL. Advances in drug therapy of cardiopulmonary arrest. *Clin Pharm* 1987;6(2):118-139.
49. Brown CG, Davis EA, Werman HA, Hamlin RL. Methoxamine versus epinephrine on regional cerebral blood flow during cardiopulmonary resuscitation. *Crit Care Med* 1987;15(7):682-686.
50. Berg RA, Otto CW, Kern KB, Sanders AB, Hilwig RW, Hansen KK, Ewy GA. High-dose epinephrine results in greater early mortality after resuscitation from prolonged cardiac arrest in pigs: a prospective, randomized study. *Crit Care Med* 1994;22(2):282-290.
51. Hoekstra JW, Van Ligen P, Neumar R, Werman HA, Anderson J, Brown CG. Effect of high dose norepinephrine versus epinephrine on cerebral and myocardial blood flow during CPR. *Resuscitation* 1990;19:227-240.
52. Callahan M, Madsen CD, Barton CW, Saunders CE, Pointer J. A randomized clinical trial of high-dose epinephrine and norepinephrine vs standard-dose epinephrine in prehospital cardiac arrest. *JAMA* 1992;268(19):2667-2672.
53. Walker GL, Williamson PM, Ravich RB, Roche J. Hypercalcaemia associated with cerebral vasospasm causing infarction. *J Neurol Neurosurg Psychiatry* 1980;43(5):464-467.
54. Stempien A, Katz AM, Messineo FC. Calcium and cardiac arrest. *Ann Intern Med* 1986;105(4):603-606.
55. Eisenberg M. The problem of sudden cardiac death. In: Eisenberg M, Berger L, Hallstrom A, eds. *Sudden cardiac death in the community*. New York: Praeger Scientific 1975:14.
56. Eisenberg M, Hallstrom A, Berger L. The ACLS score: predicting survival from out-of-hospital cardiac arrest. *JAMA* 1981;246(1):50-52.
57. Niemann J. Cardiopulmonary resuscitation in the critically ill. In: Parrillo J, ed. *Critical decisions*. Toronto: BC Decker, 1988:15.
58. Edgren E, Hedstrand U, Kelsey S, Sutton-Tyrrell K, Safar P. Glasgow-Pittsburgh coma score as predictor of neurologic outcome in comatose survivors of cardiac arrest (abstract). *Crit Care Med* 1989;17:S76.
59. Harper SJ, Wilkes RG. Posthypoxic myoclonus (the Lance-Adams syndrome) in the intensive care unit. *Anaesthesia* 1991;46(3):199-201.

Hamill Discussion

Durbin: Robin, one of the things that I think we've heard in the last few talks is that the only thing that really works in resuscitation is to perform CPR well. A few years ago the JCAHO dropped its requirement that staff physicians (attending), in fact, be trained in basic CPR. And, many institutions, our own included, do not require that the house staff, attending physicians, or specialist physicians demonstrate current competence in CPR. One of the reasons I support ACLS training for all house staff is that, in fact, part of ACLS includes competency examinations in BLS. Of course, I don't tell the hospital administration that. I believe that's the most important thing we can provide within a hospital. I think one of the reasons Virginia has such good outcome is because we require ACLS,

which includes BLS.¹ Within that critical 6-minute interval, we deliver the first components of the chain. We can provide the two things that we know work well—rapid access to CPR and then advanced life support soon after. That's what I gleaned from your presentation, as being important. Despite that, the outcome is still predetermined in at least 75% of the cases, and we're not likely to be successful. In fact, if we define a successful code as providing the resuscitative components appropriately, then it's a successful code, regardless of the patient outcome.

1. Schwenzer KJ, Smith WT, Durbin CG Jr. Selective application of cardiopulmonary resuscitation improves survival rates. *Anesth Analg* 1993; 76(3):478-484.

Hamill: That's true. I guess that my bias, though, is that even though we have a successful code, I don't think

that should be the yardstick that we use. In terms of evaluating the technology itself and any components of it, we need to look at the impact on ultimate outcome because I think that's the bigger issue.

Barnes: I just want to reinforce what you said about the futility of resuscitation efforts beyond 15 minutes. I think it's more than just the cost of the hospital staff's being there for 2 or 3 times longer than necessary. The increased probability that if you continue and the patient has a return of spontaneous circulation, stays in the hospital, but can never be discharged or must be discharged to dependent living at a tremendous expense. I think that really raises the cost.

Hamill: In terms of prolonged resuscitative efforts in the elderly, especially, a study that looked at survival

of elderly patients showed their mortality at 60-70% or so if they required more than 3 days of mechanical ventilation.¹ The 1-year survival was about 3%. So, if you end up resuscitating an elderly patient—in particular, someone who has end-stage lung disease—and they sustain a flail chest or a flail sternum and end up having to be ventilated for 3 days, just by virtue of having them on the ventilator for 3 days, you really have worsened their outcome.

1. Elpern E, Larson R, Douglass P, et al. Long-term outcomes for elderly survivors of prolonged ventilatory assistance. *Chest* 1989; 96: 1120-1124.

Aufderheide: In Milwaukee, we have retrospectively analyzed the relationship between resuscitation time and survival rate for patients with ventricular fibrillation (VF) and pulseless electrical activity (PEA), formerly termed electromechanical dissociation, or EMD. Pionkowski et al¹ demonstrated a linear drop off in the survival rate, as a function of resuscitation time, that reached a change in slope at approximately 20 minutes for ventricular fibrillation patients. Stueven, et al² attempted a similar linear regression analysis for patients in PEA and noted an increase in the successful resuscitation rates over time up to 7 minutes, and then a linear reduction in resuscitation rate from 10 to 15 minutes. Additionally, it appeared that all PEA patients who were saved had a sustaining pulse in less than 33 minutes of resuscitation time. These data support Tom Barnes' comment regarding a substantially lower possibility for successful resuscitation after about 15 or 20 minutes of CPR.

However, in the same paper, Pionkowski et al¹ also reported some patients in ventricular fibrillation who were discharged from the hospital alive after as much as 60 minutes prehospital resuscitation time. Some

PEA patients in Milwaukee achieved a transient pulse within 33 minutes but required a total of 50 minutes resuscitation time.

Those patients who are saved by resuscitation times longer than 20 minutes will not be saved in the future if CPR efforts are discontinued after 20 minutes. Do you feel that the medical and social costs of prolonged resuscitation are not justified for the relatively few patients who are saved, and what guidelines can you offer to the practicing clinician to assist in the difficult decision of terminating resuscitative efforts?

1. Pionkowski RS, Thompson BM, Gruchow HW, Aparhamian C, Darin JC. Resuscitation time in ventricular fibrillation—a prognostic indicator. *Ann Emerg Med* 1983; 12:733-738.
2. Stueven HA, Aufderheide TP, Waite Em, Mateer JR. Electromechanical dissociation: six years prehospital experience. *Resuscitation* 1989; 17:173-182.

Pepe: Just to reflect on that some more: we also looked at the Milwaukee system as well when we did a prospective validation of the criteria for terminating resuscitation.¹ The key is actually whether you get pulses back, even transiently, within that first 15-20 minute window. There are people who will transiently get pulses and if CPR is continued beyond 20 minutes some will survive. So, there is some capability there that suggests that you probably can go on. It's the people who get absolutely no pulses within—really, 20 minutes is the window—20 minutes of the onset of ACLS. The BLS component is really harder to nail down. The studies looked at 20 minutes regardless of previous arrest intervals. We had no survivors among those adults getting 20 minutes of ACLS without return of pulses. In a previous study published in *JAMA* last year,² when we prospectively tested those results against Milwaukee, Columbus, King County, Syracuse,

Richmond, and Houston again the only survivors beyond 20 minutes of all were those with refractory or persistently recurrent V-fib.^{1,2} However, if you did get any pulses back within that 20 minutes, you still had about a 25% chance of coming back. Granted, there are subcategories. For example, if you got transient pulses and lost them again, your chances were less but still there was a chance. So, I agree with you. I am very aggressive in those folks in whom we are able to get at least something back even if only transiently, but I'm as equally aggressive about calling the resuscitation if we don't get pulses back in the first 15 or 20 minutes.

1. Pepe PE, Brown CG, Bonnin MJ, et al. Prospective validation of criteria for on-scene termination of resuscitation efforts after out-of-hospital cardiac arrest. *Acad Emerg Med* 1994;1:315 (abstract).
2. Bonnin MJ, Pepe PE, Kimball KT, Clark PS. Distinct criteria for the termination of resuscitation in the out-of-hospital setting. *JAMA* Sept 22 1993; 270:1457-1462.

Aufderheide: I would like to make the point that the practicing clinician needs an established national consensus opinion and easily applied guidelines to allow practical implementation of these concepts.

Pepe: One thing I have also been very interested in recently, is the issue of waiving (not even initiating) resuscitation. There's been a great movement toward saying that if victims are asystolic, you're not going to get them back, so let's not even start resuscitation.¹ That may become a self-fulfilling prophecy in some situations. So, we actually took a look at that, in the Seattle and Houston EMS systems, and we found that if we did bring some asystole cases back it was a small percentage, like 1-2%, but we can get some of those people back.² But we also found that in those asystole cases when the arrest was unwitnessed and

no one performed bystander CPR, we didn't get anybody back, period.² So there may be some criteria we can use clinically, but in lieu of those criteria, I would aggressively start the resuscitation, unless they're livid, or are in rigor mortis, or we've got DNR orders. But at the same time, I'd just as aggressively terminate it if there is no response or pulses within the first 20 minutes or so.

1. Pepe PE, Levine RL, Fromm RE, Curka PA, Zachariah BS, Clark PS. Cardiac arrest presenting with rhythms other than ventricular fibrillation: Contribution of resuscitation efforts toward total survivorship. *Crit Care Med* 1993.
2. Pepe PE, Cobb LA, Persse DE, et al. Improved criteria for waiving resuscitation efforts for out-of-hospital cardiac arrest. *Ann Emerg Med* 1994;23:619.

Halperin: I think this 6-minute starting time is probably too long. By cutting that down to a couple of minutes, we could save a lot of lives. I don't know what the logistical problems are, but it seems like that could be shortened considerably in a hospital. Maybe the respiratory therapist could actually be a part of that.

Sanders: The point I'm concerned about is the issue of physicians writing DNR orders without patients' consent. There's no question that if the patient feels that he or she does not want to be resuscitated, then we should respect those wishes. But we need to be very cautious about physicians making those judgments unilaterally when conclusive data may not be available. You presented data on 21 patients with AIDS or 40 patients with dementia who did not survive cardiac arrest. Is that definitive data proving that resuscitation attempts are futile? There are very few conditions that have justified futility in cardiac arrest—dependent lividity, decapitation, rigor mortis, etc. But beyond these indications of death, it's the patient's choice whether resusci-

tation be attempted. The literature should be constantly monitored for new studies demonstrating futility for specific conditions—good clinical studies from multiple centers.

Hamill: You need to look at the whole picture. But I would suggest to you that CPR is a therapeutic modality, not unlike antibiotics, not unlike chemotherapy. You use your judgment in applying all of them. If a family came to you and said "I want Aunt Tilly to have chemotherapy because I read something in McCall's, that says that it's going to make a difference," you're not going to give it—because it's not indicated. Yes, it's not 100%. But I think you must take the numbers and look at the probabilities. If the patient has not one of these factors but four of them—all of which are connected to poor outcome—if you have comorbid disease plus advanced age and all the rest of it, then you need to take those into consideration. Are you doing more harm than good? Are you ultimately going to alter outcome or not? It's not an easy decision. Ideally, what I'm suggesting is that the patient be involved in those decisions whenever possible, but I think when you have someone in the process of dying, and nothing you can do will change that, then performing CPR is not appropriate. I think in those circumstances in which—for example, end-stage ARDS, where they're actually dying of hypoxia or whatever and you can't fix that—trying to resuscitate them when you can't alter the underlying process doesn't accomplish anything.

Durbin: Just a brief comment about the study you talked about.¹ We looked at patients who died with and without CPR to come up with those numbers, and what we found was that we didn't apply CPR universally to people dying in the hospital. Right now we're looking at whether DNR

orders influenced those decisions not to resuscitate. We're not quite sure what the determining variables were. We just know that we seem to be picking the right people to try to resuscitate which results in our high success rate compared to other studies of in-hospital resuscitation. I don't think it's because we're so great. I think it's because we pick the right people. I just don't know yet how we chose to resuscitate those who are good candidates and not the others.

1. Schwenzer KJ, Smith WT, Durbin CG Jr. Selective application of cardiopulmonary resuscitation improves survival rates. *Anesth Analg* 1993; 76(3):478-484.

Rubenfeld: This whole notion of defining futility as a certainty is sort of ridiculous. If our criterion for saying that something is inappropriate or that we're not going to do it, is that it's impossible, then we can all go home because there's no way that we can sit around and define what's impossible with any amount of research. Zero out of ten thousand still isn't impossible. If in the first 10,000 cases it doesn't happen, there's a 50% chance that it'll happen once in the next 10,000. I think that we need to sort of break apart what we mean by the word futility and convince ourselves that we actually don't really mean certain or impossible or inevitable. At least experimentally, we can't define any of those. It's an act of faith.

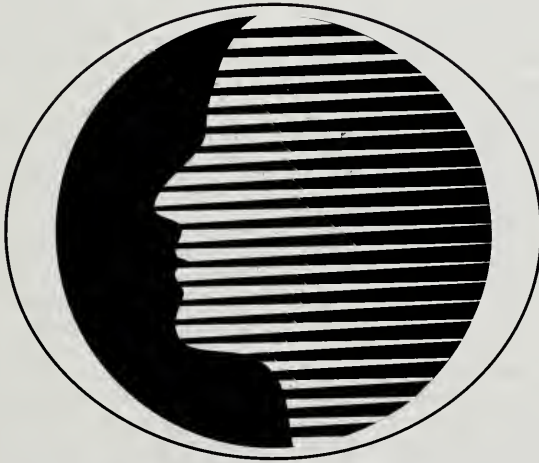
1. Sherman DA. The probability of inevitability: The inherent impossibility of validating criteria for brain death or 'irreversibility' through clinical studies. *Statistics in Medicine* 1987;6(July-Aug. 5):535-553.

Malinowski: I notice that its probably better for you to arrest on the streets of Seattle than in the ward of an acute care hospital. Would you comment on that? I know you did mention that you thought that was

probably related to the length of time that the patient had been in cardiorespiratory arrest, but I wonder if you could comment on whether you thought training the nurses and bedside providers as well as maybe Paul (Pepe) trains his fire department would make a difference.

Hamill: I don't think that's the issue. I actually think that this is one of those components that's going to become worse rather than better—as the nursing coverage lessens due to costs containment. Patients are left alone for extended periods of time. The nurse goes in to check vitals and finds

the patient dead in bed and calls a code on somebody who's been lying there for 15 minutes or 3 hours. You're often looking at a tremendous delay before resuscitation is initiated, not because somebody doesn't know what to do but because nobody was there when the arrest occurred.



FACE TO FACE WITH CHANGE

AARC 41st Annual Convention and Exhibition
December 2-5 • Orlando, Florida

Do-Not-Resuscitate Orders: A Critical Review of the Literature

Gordon D Rubenfeld MD

- I. Introduction
- II. Surveys of Health-Care Providers, Patients, & Surrogates
- III. Epidemiology of DNR Patients
- IV. The Limitations of Advance Directives
- V. The Future of DNR Orders
- VI. Summary

Introduction

In the three decades that cardiopulmonary resuscitation has been in wide use, there have been extraordinary changes in medical-care delivery, the process of dying, and the doctor-patient relationship. Our current state of practice regarding withholding and withdrawing life support has evolved by complex mechanisms involving the courts, ethics panels, physicians, and various patient-advocacy groups. Detailed discussions of the legal precedents and ethical principles that form the groundwork of current thought on terminal-care decisions can be found in reviews of these topics.^{1,2} This paper explores the extensive empirical medical literature devoted to do-not-resuscitate (DNR) orders, the process surrounding the decision to write the order, and the outcomes of patients for whom such an order is written.

Cardiopulmonary resuscitation (CPR) is the only medical therapy provided in the acute-care hospital that is administered without a physician's explicit order, but a physician's order is required to stop or prevent it from starting. Early proponents of CPR recognized that it was a therapy to be applied judiciously;³ however, prior to 1976 there was little discussion about DNR decisions. In 1965, a subtle but important transformation occurred that contributed to a new psychology.⁴ No longer a medical procedure to be applied only in selected cases by physicians,

CPR was redefined as an emergency procedure to be initiated by a broad range of personnel in many settings outside the hospital. This sense of emergency permeated the culture of CPR providers and returned with them to the hospital—a setting in which every cardiac arrest does not merit an obligatory response.

Efforts were made to limit resuscitation in clinical practice well before the acknowledgment of formal DNR orders in 1974.^{5,6} Anecdotal reports conveyed concern that consent for DNR was erratic and communication errors occurred between physicians and CPR providers. Physicians were reluctant to document DNR decisions in the medical record for fear of prosecution for failing to provide potentially beneficial emergency care. Code phrases like "tender loving care only" or symbols like purple dots on chart covers were used to indicate patients deemed inappropriate for CPR. Slow or 'sham' resuscitations—intentionally ineffectual resuscitations—were performed to give the appearance that all possible therapy was being provided.

In 1976, in the wake of the Quinlan case, a watershed series of articles was published in the *New England Journal of Medicine*. The Massachusetts General and Beth Israel Hospitals reported their guidelines for handling DNR decisions.^{7,8} They acknowledged the rights of a competent patient to refuse any medical care, the role of surrogates in making decisions for incompetent patients, the problematic nature of decisions in the absence of surrogates or expressed wishes, and the importance of an open policy regarding DNR orders. In the same issue Sissela Bok brought the "living will" to the attention of many readers' attention for the first time.⁹

During the late 1970s and early 1980s, investigators began to look closely at the outcomes of in-hospital CPR. Specific subgroups of patients with cancer, stroke, and chronic renal failure had low survival-to-hospital-discharge rates despite resuscitation. Meanwhile, states were

Dr Rubenfeld is a Robert Wood Johnson Clinical Scholar, University of California at San Francisco, San Francisco, California

A version of this paper was presented by Dr Rubenfeld during the Journal Conference, "Resuscitation in Acute Care Hospitals" held in Cancun, Quintana Roo, Mexico in October 1994.

Reports: Gordon D Rubenfeld MD, Box 0903, University of California San Francisco, 521 Parnassus Ave, San Francisco CA 94143-0903.

passing "natural death" legislation that gave statutory support to advance directives. A growing body of case law justified decisions to withhold or withdraw any medical care, in the proper setting.

With these changes in medical decision-making grew an intense interest in studying whether the goals of advance directives and formal DNR orders were being met. Does a formal DNR policy lead to better communication between physicians, patients, surrogates, and other members of the health-care team about decisions not to provide CPR? Do advance directives enhance patient autonomy by matching desired therapy with what is actually received? Do advance directives and DNR orders lead to more appropriate use of life sustaining therapy (ie, less intensive care) when it can only serve to prolong an inevitable death? The large body of research that attempts to answer these questions can be divided by methodology into the following topics: (1) surveys of health-care providers, patients, and surrogates, (2) epidemiology of DNR patients, and (3) the limitations of advance directives.

Surveys of Health-Care Providers, Patients, & Surrogates

When outpatients were asked whether they would like to discuss life-sustaining therapy, nearly 70% said they would. Patients feel empowered by discussing these issues and wish their physicians would initiate conversation on this topic.¹⁰ A later study found that more than 90% of outpatients and the general public were interested in having either an advance directive or a health-care proxy.¹¹ Despite this enormous interest, very few individuals actually write advance directives. Fewer than half ever explicitly discuss their wishes with family members.¹² Respondents cite lack of physician initiative as an important barrier to their giving advance directives.^{10,11} These data, along with the observation that interested individuals are indistinguishable by demographic criteria from those who are not interested, have been used to encourage health-care providers to approach *all* patients about end-of-life decisions.

Surveys have provided information on patients' preferences for therapy in various scenarios. In general, patients prefer comfort measures to life prolongation when faced with terminal illness or irreversible coma. Over 80% of patients say they would refuse nutrition, antibiotics, or mechanical ventilation if they were in a persistent vegetative state or were terminally ill with dementia.¹¹ However, there is an important minority in all surveys who prefer aggressive therapy regardless of the scenario. Seventy-eight percent of patients who agreed with the statement "I do not want my life prolonged, and I do not want life-sustaining treatment to be provided or continued if the burdens of the

treatment outweigh the expected benefits" wanted CPR in the event of a cardiac arrest.¹³ When faced with the scenario of terminal illness defined as "dying with a disease that could not get better no matter what your doctor does", 34% wanted CPR and 27% wanted admission to an intensive care unit.¹⁴ Twenty percent of nursing home residents said they wanted CPR, mechanical ventilation, and hospitalization even if they were terminally ill or permanently unconscious.¹⁵

An important concern for those faced with interpreting advance directives is whether the stated choices are stable over time. A patient who filled out an advance directive 3 years ago may no longer agree with its contents but be unable to communicate his change-of-heart when admitted to the hospital. Overall, patients do change their attitudes regarding end-of-life care. Patients with more education, who are black, and who have less social support tend most to change.¹⁶ Initial choices for less care, having an advance directive, and repeated discussions over time all make stable decisions more likely.^{16,17} Of note, among patients who initially choose the least care, subsequent decisions for more care are unlikely.¹⁶

For advance directives and health-care proxies to fulfill their goals of enhancing patient autonomy, we must be confident that actions based on them approximate the decisions the patient would have made. Proving this hypothesis would be difficult because there is no way to ascertain what an individual patient would *really* choose in the clinical settings in which advance directives are enacted. A number of studies have attempted to ascertain the extent to which a patient's choices agree with those of a surrogate, using clinical vignettes. The surrogates (physicians, nurses, and family members) did little better than chance at selecting the therapy that patients chose.¹⁸ Physicians tend to underestimate patients' desires for resuscitation whereas family members overestimate it. This observation has been used to advocate a procedure-specific advance directive that provides a menu of therapies, given various scenarios. Studies of agreement over therapy have not been repeated using procedure-specific statements, so the advantages remain untested. Clinical vignette research has raised substantial doubts as to whether health-care surrogates are able to choose the same therapies that the patient might have chosen had he been given the opportunity.

The diversity of responses regarding treatment choices based on surveys and vignette research can be due to several factors. To some extent, it represents true differences in the values people assign to various outcomes. However, hypothetical vignettes are likely to fail to represent actual behavior for several reasons. Written scenarios cannot capture the reality of clinical situations and, therefore, may not elicit responses that correspond to those that might emerge at the

bedside. These studies provided only cursory information about outcomes, so patients and their family members were left with whatever individual preconceptions they had about CPR effectiveness. Finally, closed-choice surveys are a poor instrument to ascertain attitudes in a situation that is bounded by uncertainty. Respondents may be more willing to 'gamble' on CPR if the negative outcomes—prolonged hospital survival followed by death or resuscitation to a debilitated state—are not made explicit.¹⁹

Both physicians and lay persons are influenced by social factors that have little impact on assessing prognosis or benefit from CPR when they decide on the appropriateness of resuscitation. Patients with acquired immune-deficiency syndrome and cancer are much more likely than their equally ill counterparts with cirrhosis and heart failure to be approached about and consent to a DNR order.²⁰ When faced with the decision to withdraw therapy, physicians are less likely to do so if the patient's illness is iatrogenic or unexplained, even when these factors are not associated with a different prognosis.²¹ Judgments of non-physicians are also affected by social factors that are of questionable significance in the decision to offer CPR. In one study cancer, alcoholism, substance abuse, and physical handicaps all decreased the likelihood that resuscitation would be offered to hypothetical patients with similar prognoses.¹²

One survey of a broad range of health-care professionals has yielded particularly disturbing results.²² A strong sentiment that too much care is provided to the terminally ill emerges from the study respondents. Sixty-seven percent felt that mechanical ventilation was used inappropriately in the care of dying patients. Similar data were obtained on CPR and facilitated nutrition. The same survey also revealed a striking lack of knowledge or acceptance of modern ethical and legal principles. Over time, a broad consensus has developed that distinctions between ordinary and extraordinary care and withholding and withdrawing care are more confusing than enlightening in medical decision-making.² However, a majority of responding health-care professionals believed that withdrawing a treatment was different than withholding it and three quarters believed there was a distinction between ordinary and extraordinary care. Confirming this misconception, 47% felt that food and water should not be discontinued even when other forms of life support are stopped. Imposing these distinctions may lead to unwanted care and unnecessary ethical dilemmas.

Survey research has been invaluable in breaking down the barrier of silence that has prevented clinicians from approaching patients about advance directives. Most people would prefer comfort measures to CPR and other life-sustaining therapies, when faced with terminal illness. This research has also uncovered difficulties in the application

of surrogates' judgments that may not represent patients' choices. Finally, health-care professionals believe that they provide inappropriately excessive care to the dying yet are confused about important ethical concepts that might lead to more appropriate care.

Epidemiology of DNR Patients

This body of literature is difficult to synthesize given the wide variability in study design, populations, and rapid secular trends in practice during the 1980s. However, these studies provide a series of snapshots that outline the consent process, describe the typical DNR patient, explore the practical therapeutic implications of a DNR order, and reveal international differences in thinking about end-of-life care. It is important to note that all these data were collected prior to the Patient Self Determination Act (PSDA) of 1991. This legislation requires that all patients receive information about advance directives at the time of hospital admission.

DNR orders are written in 3% of patients admitted to the hospital²³⁻²⁶ and, from more recent data, in 9% of ICU patients.²⁷ Among patients who die in the hospital, 70-84% have a DNR order on record.²³⁻²⁸ One ICU reported that 97% of the patients who died in the unit had DNR orders written in their charts.²⁹ A more representative national sample suggests that 70% of ICU deaths are preceded by a DNR order.²⁷

The institution of DNR policies has led to a noticeable increase in the documentation of the rationale for withholding CPR. In the majority of cases (56-86%), this is done after admission to the hospital and in consultation with the family.^{23,25,30,31} Patients were able to participate in only 14-28% of DNR decisions.^{23,30,31} DNR orders in the absence of surrogates or against the requests of surrogates occur in only a few percent of all cases. One study documented a few cases of unacceptable informal orally communicated DNR orders used to circumvent a resistant family's demands for CPR.²⁴

An important goal of formal DNR orders is to improve communication about instituting CPR. DNR orders are intended to facilitate and document the decision to withhold CPR and to convey this information to caregivers responsible for initiating such care. Obviously, there is a broad range of patients in whom a DNR order is appropriate. Contrast 3 hypothetical DNR cases: (1) A 62-year-old man with emphysema was admitted for pneumonia. He had written an advance directive that stated he did not want CPR but has been successfully extubated after prolonged mechanical ventilation twice in the past. (2) A 78-year-old woman with severe dementia who has a living will was transferred from the nursing home with urosepsis. (3) A 37-year-old trauma patient with severe closed-head injury

and acute respiratory distress syndrome was in the ICU with progressive organ failure, inadequate oxygenation on FDO_2 1.0, and hypotension requiring high-dose vasopressors. All 3 cases might show up in a series of DNR patients, but they have little else in common aside from their DNR status. The level of intensive care each would receive, the treatments that might be withdrawn or withheld, and the timing from admission to the DNR order would vary widely in each case.

In fact, there is considerable variation in the aggressiveness of care patients receive after a DNR order.^{23,32,33} One study that used blinded chart reviews by expert physicians found only a few cases in which care was deemed inappropriately excessive after the DNR order.³⁴ Physicians have different perceptions of just what the "resuscitate" in do-not-resuscitate means. Although there was general agreement that chest compressions and cardioversion were "resuscitation," physicians disagreed on whether new antiarrhythmic or vasoactive drugs could be started in a patient with a DNR order.³² Physicians providing on-call coverage for a DNR patient, called in the middle of the night, may not share the same concept of resuscitation as that negotiated between the patient's primary physician and family.³⁵ Again there was general agreement that CPR, cardiac pacing, and mechanical ventilation should be withheld, but there was greater uncertainty with regard to ICU transfer, vasopressors, and blood transfusions. Some institutions have responded to this phenomenon with procedure-specific DNR orders that list which aspects of resuscitation are to be withheld.³⁶ Unfortunately, an instrument meant to convey detailed information to colleagues may overwhelm a surrogate and lead to choices that are medically irrational. There may be situations in which it is reasonable to choose some components of CPR but refuse others. For example, patients with end-stage lung disease might want to be cardioverted for sustained ventricular tachycardia but not intubated. However, patients should understand that the intervention, CPR, as it is applied to the unresponsive, apneic, or pulseless patient is a package deal. Chest compressions for ventricular fibrillation without electrical defibrillation or epinephrine without oxygenation are even less likely to be effective than the data already suggest for in-hospital resuscitation. The rationale behind limited-code requests should be explored for hidden misconceptions. Patients may feel that some aspects of resuscitation are more or less heroic than others, or they may not understand that the majority of patients require prolonged mechanical ventilation and ICU care after in-hospital resuscitation.

The United States has generated much of the world's medical, ethical, and legal literature on DNR orders. However, there is a limited published experience from colleagues abroad that is illuminating. A critical care group

from Belgium stated in an article reporting on terminal events in their ICU:³⁷

Of particular importance is the primary role of the physician in these ultimate decisions. Although the patient and his family are regularly consulted about their feelings and desires, the decision not to perform CPR or to discontinue therapy is ultimately made by the ICU staff.

An article published in 1991 from a district general hospital in England showed the consequences of not having a formal DNR policy. Orders in the chart were cryptic and haphazardly documented. Communication was so poor that nurses claimed they would not call the "crash team" on 40% of the patients designated by doctors as having full-code status; but would call the team for one third of the patients whom doctors had designated as having DNR status.³⁸ Physicians in Sweden, who also practice in the absence of a formal DNR policy, only document 18% of their DNR orders in the medical record. The majority were written, frequently in coded language like "0 action" or coded symbols (a heart with a line through it) in the nursing care plan with pencil. Seventy percent said they never or rarely discussed DNR decisions with family members and 90% never or rarely discussed the decision with the patient.³⁹ Clearly, the approach to end-of-life decision-making in the United States is markedly different from other developed countries. It is not clear whether recent interest in unilateral DNRs and medical futility are moving us closer to the European view or whether burgeoning consumerism will cause the Europeans' medical decision-making to become more like ours.⁴⁰⁻⁴²

The Limitations of Advance Directives

The goal of advance directives is to allow individuals to influence choices made on their behalf by projecting their autonomous decisions on future events. For these projected autonomous decisions to be valid and effective, they must overcome a series of impediments (Table 1). Additional information about CPR outcomes, particularly if provided in a creative way, can influence patients' choices for CPR.⁴³ We have already discussed how some patients' opinions about care can be unstable over time. Even under optimal study conditions, advance directives may only be available for a third of hospitalized patients.¹⁵ Finally, those involved with the decision must be able to apply previous statements at the bedside and turn them into practical decisions to withhold or withdraw specific therapies.

Three prospective studies suggest that advance directives, for unknown reasons, have little impact on subsequent care. In an important study, Danis and colleagues¹⁵

Table 1. Prerequisites for Effective Projected Autonomous Decisions.

Information
Choices should be informed, including an understanding of the likely outcomes and burdens of CPR and intensive care in various situations.
Communication
Personal choices kept private can have little impact at the bedside.
Stability
Informed choices that were communicated in the past should be similar to the choices that the patient would have made today.
Availability
The instrument for communicating the stable, informed choice must be available to physicians when decisions need to be made. If surrogates are absent and the advance directive is at the nursing home projected autonomy is impossible.
Applicability
The interaction between physician and document or physician and surrogate must lead to some change in therapy. This requires that the decision makers be able to interpret previous statements and apply them at the bedside allowing for an infinite number of potential clinical scenarios. Presumably, in some cases, the management is different from what would have occurred in the absence of an advance directive.

demonstrated that, in a quarter of the relevant events, medical care was inconsistent with previously stated requests. Advance directives were actually present more often in the inconsistent-care cases. From the events described in the article, it is difficult to know whether there was really a lapse in appropriate decision-making. For example, one patient who had requested not to be placed on a ventilator was briefly intubated because of a seizure; and a patient with end-stage cardiomyopathy who had requested hospitalization in his directive was not hospitalized when he became incompetent and too severely ill to benefit from more aggressive care. These events exemplify how difficult it can be to turn advance directives into specific actions at the bedside. Even if a detailed, procedure-specific document is used, as it was in the Danis et al¹⁵ study, it is impossible to anticipate all clinical scenarios. Furthermore, many patients acknowledge the importance of affording their physicians the flexibility to make bedside adjustments to previously stated wishes.⁴⁴

Schneiderman, in a randomized trial, found no difference in the percent of patients receiving CPR, dying at home, or any of a series of variables measuring resource consumption among those offered an advance directive, those who had written an advance directive, and a control group.⁴⁵ Recent data from the Study to Understand Prognoses and Preferences for Outcomes and Risks of Treatments (SUPPORT) corroborates these findings.⁴⁶ In a cohort of patients

with a 45% 6-month mortality, 20% had an advance directive and only 6% of these were mentioned in the patient's medical records. There were no differences between the care and resource consumption of those who had advance directives and those who did not. Both groups were equally likely (about 13%) to receive CPR prior to death.

These recent articles present a troubling counterpoint to our national focus on advance directives. There is no way, from currently available data, to tell which of the hurdles listed in Table 1 prevent advance directives from being effective. It may be the case that everyone, regardless of whether they have an advance directive, is receiving compassionate care consistent with prior wishes. If so, then it is unclear exactly what role the advance directive plays in the process. Certainly some impediments to the effectiveness of advance directives have been addressed by the PSDA. Patient education, discussions with physicians and surrogates, and availability of advance directives may be improved by a heightened awareness created by efforts to comply with the new law. Studying the effects of any changes in the DNR process is hampered by the lack of an external standard for judging whether any particular decision has fulfilled the goals of projected autonomy.

One goal for which there is an easily measured external standard is hospital charges. Greater use of advance directives and less aggressive care at the end of life has been advocated as a source for cost reduction in a health-care system that is increasingly cost-sensitive. The prospective studies cited above found no difference in resource utilization related to the presence of an advance directive. Two retrospective studies had opposite findings and showed large differences in length of stay and hospital charges.^{47,48} Both used a similar research strategy: they collected a list of all hospital deaths, reviewed the charts for evidence of either a DNR order or the presence of a discussion about an advance directive, totaled the hospital days, and computed the charges for care. Patients who died with a DNR order written before admission had significantly shorter hospital stays and charges than those without DNR orders. Patients with DNR orders written during the hospitalization incurred the highest charges and longest hospitalizations (Table 2). The other study found that patients who died with an advance directive documented in the chart within 48 hours of admission had one quarter the median hospital charges of those without such documentation.⁴⁸

These studies demonstrate that if a decision to do less can be made early in the course of illness, then less is done. Unfortunately, neither retrospective study has shown whether advance directives or preadmission discussions can facilitate this decision. Patients who lack a *documented* advance directive may have had a directive or discussion that the physician and/or surrogates chose to ignore on a given hospital admission. Maksoud and his col-

Table 2. Length of Hospital Stay and Charges by DNR Status*

	Number of Patients (%)	Median LOS† (days)	Median Charges (\$)
Before admission	107(13)	4	5,504
None	227(27)	6	32,585
After admission	512(60)	14	40,342

* Adapted from Reference 47, with permission

† LOS, length of hospital stay

leagues⁴⁷ do not say exactly how they ascertained prehospital DNR status. If it was obtained by chart review, like the rest of the data from their article, then it is subject to the same documentation bias as the study by Chambers et al.⁴⁸ Physicians may be more likely to document advance directives that they know about and have decided to follow. Therefore, we cannot tell whether it is the advance directive that caused the lower resource use or an early decision to use fewer resources that prompted physicians to document and follow advance directives.

The decision to withhold and withdraw care may be difficult and delayed for a variety of reasons. There may be uncertainty in the patient's prognosis, particularly with regard to neurologic outcome. There may be agreement about outcome but a grieving family may not be prepared to accept that their loved one has reached the decision point. Surrogates may accept the physician's prognosis and still want to risk any chance for survival no matter how small. There may be disagreement among family members about the intent of a patient's prior wishes regarding health care. Clearly, a piece of paper appointing a health-care proxy or listing desired therapies in hypothetical scenarios can not be expected to resolve all of these deeply rooted conflicts.

The Future of DNR Orders

The next decade of DNR decision research will try to reclaim CPR as a medical procedure for appropriate cases rather than an emergency procedure to be attempted in all cardiac arrests. Defining "appropriate," like "futility," or "medically necessary" is a challenge both for society and medical practitioners. The simplest of such cases are those in which patient, family, and physician have already agreed that CPR is to be withheld when the patient is discharged from the hospital. There can be a no more tragic scenario than that involving a terminally ill patient who is prepared to die but whose family, alone and stressed, calls the ubiquitous 9-1-1 for help only to find that they have unleashed a whirlwind of medical care. Local Emergency Medical Systems, physicians, and hospice centers should develop coordinated care plans so that families can have

rapid intensive support that does not include obligatory CPR when loved ones are dying at home.⁴⁹

Cases in which a miniscule potential benefit of CPR and intensive care is outweighed by the burdens they impose present a more intractable problem. As data mount on the costs of CPR and abysmal quality-of-life outcomes in specific populations,⁵⁰ it is increasingly difficult for clinicians to ethically provide and for society to pay for such care. One way to reach consensus on who these patients are is to do so at the community level. Broad coalitions in cities like Denver, Colorado and Santa Monica, California are convening to develop lists of conditions for which patients will not receive CPR or other intensive care.^{42,51} This community based definition of "futility" avoids the accusation that a cabal of physicians or payors are conspiring to limit care for their own ends. If such systems are applied equally to all residents of a community they avoid the criticisms of economic and racial bias. It is still too early to tell how much authority these guidelines have or what might happen to those who persist in providing excessive care to patients on the list.

Practitioners of critical care may be their own worst enemy when it comes to discussing end-of-life decisions with families. Over the years we have done such a good job promoting CPR, intensive care, and the technologic marvels that accompany them, that we may have actually oversold these therapies. Not only the general public, but also many of our colleagues whose patients become critically ill, have high expectations of us. Now is the time for organizations with an interest in the appropriate use of CPR, like the American Heart Association and the American Association for Respiratory Care, to invest in patient and physician education. For truly informed consent to be obtained and utilization of CPR to be appropriate, providers and consumers must be educated about the outcomes and costs of this care. Perhaps patients need to have a more realistic view of CPR and its consequences. When patients are approached about writing an advance directive, perhaps they should view a film of a real resuscitation and be given to understand how unlikely a good outcome is, for given conditions. Health-care providers who advise patients about advance directives should be made aware of hospital CPR outcomes data during their Advanced Cardiac Life Support recertification classes. Finally, research should continue to investigate whether new formats of advance directives that are tailored to individual ethnic, educational, and religious backgrounds can improve the effectiveness of projected autonomous decisions.

Summary

Formal DNR policies have improved the process by which decisions to withhold CPR are made. Slow and sham

codes are now rare. Communication between physicians and patients and between physicians and providers of in-hospital CPR clearly exceeds that in other developed countries. Results of early studies that examined the care received by those with and without advance directives have been unable to document differences. A more realistic appraisal of the potential role for advance directives coupled with an emphasis on education and rational treatment guidelines should promote continued improvement in the compassionate use of cardiopulmonary resuscitation.

REFERENCES

- Office of Technology Assessment. Life-sustaining technologies and the elderly. Philadelphia: Lippincott, 1988.
- President's Commission for the Study of Ethical Problems in Medicine and Biomedical and Behavioral Research. Deciding to forego life-sustaining treatment: a report on the ethical, medical, and legal issues in treatment decisions. Washington, DC: 1983.
- American Heart Association Committees on Emergency Cardiac Care and Subcommittees. Standards for cardiopulmonary resuscitation (CPR) and emergency cardiac care (ECC). V. Medico-legal considerations and recommendations. *JAMA* 1974; 227(7, Suppl):864-868.
- American Heart Association, American Red Cross, Industrial Medical Association, The U.S. Public Health Service. The closed-chest method of cardiopulmonary resuscitation—revised statement (editorial). *Circulation* 1965; 31(5):641-643.
- Shey HH. Prevention of irrational resuscitation (editorial). *JAMA* 1972; 222(13):1653.
- Andree RA, Pilchik EE. When death is inexorable (editorial). *Science* 1970; 169(947):717.
- Optimum care for hopelessly ill patients. A report of the clinical care committee of the Massachusetts General Hospital. *N Engl J Med* 1976; 295(7):362-364.
- Rabkin MT, Gillerman G and Rice NR. Orders not to resuscitate. *N Engl J Med* 1976; 295(7):364-366.
- Bok S. Personal directions for care at the end of life. *N Engl J Med* 1976; 295(7):367-369.
- Lo B, McLeod GA and Saika G. Patient attitudes to discussing life-sustaining treatment. *Arch Intern Med* 1986; 146(8):1613-1615.
- Emanuel LL, Barry MJ, Stoeckle JD, Ettelson LM, Emanuel EJ. Advance directives for medical care—a case for greater use. *N Engl J Med* 1991; 324(13):889-895.
- Ebell MH, Smith MA, Seifert KG, Polsinelli K. The do-not-resuscitate order: outpatient experience and decision-making preferences. *J Fam Pract* 1990; 31(6):630-636.
- Schneiderman LJ, Pearlman RA, Kaplan RM, Anderson JP, Rosenberg EM. Relationship of general advance directive instructions to specific life-sustaining treatment preferences in patients with serious illness. *Arch Intern Med* 1992; 152(10):2114-2122.
- Garrett JM, Harris RP, Norburn JK, Patrick DL, Danis M. Life-sustaining treatments during terminal illness: who wants what? *J Gen Intern Med* 1993; 8(7):361-368.
- Danis M, Southerland LJ, Garrett JM, Smith JA, Hielemma F, Pickard CG, Egner DM, Patrick DL. A prospective study of advance directives for life-sustaining care. *N Engl J Med* 1991; 324(13):882-888.
- Danis M, Garrett J, Harris R, Patrick DL. Stability of choices about life-sustaining treatments. *Ann Intern Med* 1994; 120(7):567-573.
- Emanuel LL, Emanuel EJ, Stoeckle JD, Hummel LR, Barry MJ. Advance directives. Stability of patients' treatment choices. *Arch Intern Med* 1994; 154(2):209-217.
- Uhlmann RF, Pearlman RA, Cain KC. Physicians' and spouses' predictions of elderly patients' resuscitation preferences. *J Gerontol* 1988; 43(5):M115-121.
- Patrick DL, Starks HE, Cain KC, Uhlmann RF, Pearlman RA. Measuring preferences for health states worse than death. *Med Decis Making* 1994; 14(1):9-18.
- Wachter RM, Luce JM, Hearst N, Lo B. Decisions about resuscitation: inequities among patients with different diseases but similar prognoses. *Ann Intern Med* 1989; 111(6):525-532.
- Christakis NA, Asch, DA. Biases in how physicians choose to withdraw life support. *Lancet* 1993; 342(8872):642-646.
- Solomon MZ, O'Donnell L, Jennings B, Guilfooy V, Wolf SM, Nolan K, et al. Decisions near the end of life: professional views on life-sustaining treatments. *Am J Public Health* 1993; 83(1):14-23.
- Bedell SE, Pelle D, Maher PL, Cleary, PD. Do-not-resuscitate orders for critically ill patients in the hospital. How are they used and what is their impact? *JAMA* 1986; 256(2):233-237.
- Lo B, Saika G, Strull W, Thomas E, Showstack J. 'Do not resuscitate' decisions. A prospective study at three teaching hospitals. *Arch Intern Med* 1985; 145(6):1115-1117.
- Uhlmann RF, McDonald WJ, Inui TS. Epidemiology of no-code orders in an academic hospital. *West J Med* 1984; 140(1):114-116.
- Lipton HL. Do-not-resuscitate decisions in a community hospital. Incidence, implications, and outcomes. *JAMA* 1986; 256(9):1164-1169.
- Jayes RL, Zimmerman JE, Wagner DP, Draper EA, Knaus WA. Do-not-resuscitate orders in intensive care units. Current practices and recent changes. *JAMA* 1993; 270(18):2213-2217.
- Stolman CJ, Gregory JJ, Dunn D, Ripley B. Evaluation of the do not resuscitate orders at a community hospital. *Arch Intern Med* 1989; 149(8):1851-1856.
- Stern SG, Orlovski JP. DNR or CPR—the choice is ours. *Crit Care Med* 1992; 20(9):1263-1272.
- Evans AL, Brody BA. The do-not-resuscitate order in teaching hospitals. *JAMA* 1985; 253(15):2236-2239.
- Jonsson PV, McNamee M, Campion EW. The 'Do not resuscitate' order. A profile of its changing use. *Arch Intern Med* 1988; 148(11):2373-2375.
- La Puma J, Silverstein MD, Stocking CB, Roland D, Siegler M. Life-sustaining treatment. A prospective study of patients with DNR orders in a teaching hospital. *Arch Intern Med* 1988; 148(10):2193-2198.
- Lipton HL. Physicians' do-not-resuscitate decisions and documentation in a community hospital. *Qrb Qual Rev Bull* 1989; 15(4):108-113.
- Lipton HL. Do-not-resuscitate decisions in a community hospital: implications for quality care. *Qrb Qual Rev Bull* 1987; 13(7):226-231.
- Uhlmann RF, Cassel CK, McDonald WJ. Some treatment-withholding implications of no-code orders in an academic hospital. *Crit Care Med* 1984; 12(10):879-881.
- Mittelberger JA, Lo B, Martin D, Uhlmann RF. Impact of a procedure-specific do not resuscitate order form on documentation of do not resuscitate orders. *Arch Intern Med* 1993; 153(2):228-232.
- Vincent JL, Parquier JN, Preiser JC, Brimiouille S, Kahn RJ. Terminal events in the intensive care unit: review of 258 fatal cases in one year. *Crit Care Med* 1989; 17(6):530-533.
- Aarons EJ, Beeching NJ. Survey of "do not resuscitate" orders in a district general hospital. *BMJ* 1991; 303(6816):1504-1506.
- Asplund K, Britton M. Do-not-resuscitate orders in Swedish medical wards. *J Intern Med* 1990; 228(2):139-145.

40. Murphy DJ, Finucane TE. New do-not-resuscitate policies. A first step in cost control. *Arch Intern Med* 1993; 153(14):1641-1648.
41. Marsh FH, Staver A. Physician authority for unilateral DNR orders. *J Leg Med* 1991; 12(2):115-165.
42. Hicks L. Life or death: Health officials are pioneering broad guidelines to prevent 'futile care' in local hospitals. *Denver Business Journal* 1993; 45(4):1.
43. Murphy DJ, Burrows D, Santilli S, Kemp AW, Tenner S, Kreling B, Teno J. The influence of the probability of survival on patients' preferences regarding cardiopulmonary resuscitation. *N Engl J Med* 1994; 330(8):545-549.
44. Sehgal A, Galbraith A, Chesney M, Schoenfeld P, Charles G, Lo B. How strictly do dialysis patients want their advance directives followed? *JAMA* 1992; 267(1):59-63.
45. Schneiderman LJ, Kronick R, Kaplan RM, Anderson JP, Langer RD. Effects of offering advance directives on medical treatments and costs. *Ann Intern Med* 1992; 117(7):599-606.
46. Teno JM, Lynn J, Phillips RS, Murphy D, Youngner SJ, Bellamy P, et al. Do formal advance directives affect resuscitation decisions and the use of resources for seriously ill patients? SUPPORT Investigators. Study to Understand Prognoses and Preferences for Outcomes and Risks of Treatments. *J Clin Ethics* 1994; 5(1):23-30.
47. Maksoud A, Jahnigen DW, Skibinski CI. Do not resuscitate orders and the cost of death. *Arch Intern Med* 1993; 153(10):1249-1253.
48. Chambers CV, Diamond JJ, Perkel RL, Lasch LA. Relationship of advance directives to hospital charges in a Medicare population. *Arch Intern Med* 1994; 154(5):541-547.
49. Sachs GA, Miles SH, Levin RA. Limiting resuscitation: emerging policy in the emergency medical system. *Ann Intern Med* 1991; 114(2):151-154.
50. Rosemurgy AS, Norris PA, Olson SM, Hurst JM, Albrink MH. Prehospital traumatic cardiac arrest: the cost of futility. *J Trauma* 1993; 35(3):468-74.
51. Hudson T. Are futile-care policies the answer? Providers struggle with decisions for patients near the end of life. *Hosp Health Netw* 1994; 68(4):26-30, 32.

Rubenfeld Discussion

Hamill: What is your solution if advance directives don't have an impact? What solution would you suggest as an alternative?

Rubenfeld: The provocative answer to your question is, Who cares? If advance directives don't affect therapy, then what we really need to do is focus on educating consumers (future decision makers) and physicians about the dismal outcomes in some classes of patients following CPR. The safer answer to your question is that we need to study CPR decisions closely to discover which of the five impediments to projected autonomous decisions [refer to Table 5 in the paper] is operating. So, we need to study the process and find out where it's breaking down.

Hamill: An alternative is to assign a medical power of attorney—perhaps, not to a family member but to someone with whom the individual can discuss in detail what his or her expectations are.

Aufderheide: As an emergency physician, I am frequently forced to medically intervene because of the failure of the primary care clinician to

ever address the issue with their patients and because of their failure to communicate the patient's DNR status during emergency circumstances. From my perspective, there are many links in the DNR chain that easily and quickly break down when stressed.

Rubenfeld: I think what we need to do is to get clinicians like yourself, who have dozens and dozens of war stories, together with directors of EMS systems and directors of nursing homes and skilled nursing facilities. This seems to me to be a surmountable problem for smart people. It's a paperwork problem. It's a communication problem. It seems to me that now, particularly, with financial interest in this phenomenon increasing, there'll be funds available to prevent it from happening. So, putting together a stronger "chain of nonsurvival" makes good sense. Alternatively, we should include a DNR link in the ACLS chain of survival. This may take away from the aura of emergency response.

Aufderheide: As you mentioned, this is not just a problem with outpatients. A case in Wisconsin underscores the importance of communicating DNR orders in the hospital also. A patient was admitted to the hospital with a

DNR order written, and it was not well communicated to the staff. The patient arrested and was successfully resuscitated. He then spent the next year and a half of his life suing the institution for the pain and suffering of that additional year and a half.

Rubenfeld: You can imagine how often that happens in England. From the data I showed you, the communication among patients, physicians who write DNR orders, and the nurses who carry them out is very poor. Given the documentation that occurs in the U.S., I suspect the phenomenon you cite is relatively rare.

Boudin: Rather than putting the burden of responsibility on the primary-care physician for educating the patients about advance directives, shouldn't the hospitals have some type of obligation to provide this information?

Rubenfeld: Now they have a legal obligation. Right?—the Patient Self Determination Act. Although I'm not familiar with all its arcane details, it does require hospitals to provide some information in a brochure and to tell patients that it's available. The data that are available now,¹ which are very recent and preliminary, sug-

gest that the PSDA has had little effect at all on the number of patients who write formal advance directives. It increases the discussion between patients and surrogates, which may be a good thing. But we don't even know that, right?

I think it's the obligation of groups like this and for ACLS/BLS course designers to incorporate available CPR outcome data into their course curricula.

1. Emanuel EJ, Weinberg DS, Gonin R, Hummel LR, Emanuel LL. How well is the Patient Self-Determination Act working?: early assessment. *Am J Med* 1993;95(6):619-628.

Sanders: I think one of the problems with providing outcome data is the variability, depending on external factors. For example, if I'm having a myocardial infarction in the coronary care unit, and go into ventricular fibrillation, the chances of my being resuscitated, without even pumping on my chest, is probably 90+ %. Whereas, if I'm in the middle of Chicago, it might be 1%. If I'm in the middle of Seattle, it might be 43%. Yet these divergent outcomes are all lumped together when CPR is assessed. Telling a patient that there is a 10% chance of resuscitation may only be true under some conditions. In addition, there is very little reliable predictive data on neurologic outcome following cardiac arrest.

Rubenfeld: It is difficult. Let me just answer your question by circumventing some of it. This business about not being able to predict for individuals doesn't make sense because we predict for individuals all the time in medicine. Right? Every time we make a decision about giving thrombolytics, we're basing it on population data from a clinical trial. So, we certainly can provide patients with information. But, your point is that we don't know all of the variables that should go into the prediction. Just as it would be silly,

to quote the chance of it raining anywhere in the world tomorrow as the chance of it raining in Cancun, it would also be silly to quote to a patient the chance of survival from in-hospital CPR that applied to a whole group of hospital patients. It is a reasonable guess to provide someone with some individual numbers, based on his or her age and comorbidities. Those data do exist and are fairly consistent—*fairly* consistent. This is what we have. The alternative to that is saying, "You're going to have an arrest, and we have no idea whether you're going to survive or not," which is wrong. I think we do have outcome data. I think we need more of it; we need to know what the real predictors are.

Halperin: It seems that we may be trying to apply a different standard to CPR than we are to other medical procedures. I'm a CCU attending, and I send patients to surgery all the time. When I talk to the surgeons about any one patient, if there's only a 20% chance of survival from surgery, they usually won't do it. In CPR, where it's just as morbid and can commit the patient to as much expenses, we think 20% is actually probably pretty good, and here we are arguing over, well, "Should we not do it if it's 1% or 1/2%?" So, I'm not certain how those things reconcile. One other point—the survival rates in ICUs have skyrocketed since DNR orders have been implemented. It's not clear from your presentation how that really fits in with these outcomes.

Rubenfeld: Number one is your issue about CPR versus other medical procedures, and I think it's very important and very interesting. That's why I tried, up front, to point out where I think that transition occurred—in 1965, when CPR was actually separated out from other medical procedures. It *is* different. It's frequently provided by nonphysicians. It's the only medical therapy

that generally requires a physician to stop it. I agree with you that it's this sort of inconsistency that we need to focus on, but there are some psychological differences between the surgeon, who decides to offer a therapy to a patient, then has them literally die in her hands. I'm not sure that psychological difference is enough to ... I mean what you point to is something we need to think about. We need to think about why we approach the two therapies differently. I think there are some inconsistencies, and I'm agreeing with you. Your other point?

Halperin: It's just about the DNR orders increasing survival rates in ICUs.

Rubenfeld: Well, I think that's the same reason why the survival's better at the University of Virginia. We're picking out a better population of patients, and I think that is one of the great successes of DNR orders.

Kaye: As you've outlined, a major problem is that the practicing physician really is unaware of the many papers that address predictors of poor outcome. I think that the Heart Association has a responsibility to review the outcome literature and give some direction. All ACLS guidelines include many recommendations that they label as Class IIb, "acceptable, possibly helpful" and I think that they can look at the outcome literature in the same way. The practicing physician needs to be provided with that information so that he can make a rational decision. I think that's the responsibility of the Heart Association, and I think that clearly should be done.

Rubenfeld: Nothing would make me happier. If that information could be part of the ACLS syllabus.

Kaye: Make them Class IIb recommendations, if you like!

Sanders: The American Heart Association has provided criteria for assessing futility and has reached consensus on some conditions. For example, metastatic cancer is listed as a condition with a poor prognosis for resuscitation from cardiac arrest. Unfortunately, prognostic data are not available on many diseases because good clinical studies have not been done.

Sanders: You can suggest other things—AIDS with 2 episodes of PCP, for example. The evidence from clinical studies can be presented to a consensus panel of experts. The AHA provides the framework and guidelines for assessing scientific evidence and achieving consensus. This, as

we've stated previously, cannot be done on an individual basis where bias becomes an important factor.

Kaye: The Heart Association has only begun developing consensus on outcome predictors. They need to go much faster.

Rubenfeld: But the Heart Association has convened panels; it has done that. Now, you may not agree with everything they say, or it may need updating. It's been a couple of years, but it is an active process.

Pepe: One of the problems that I see is that it is, and must be, a multifactorial prediction formula. Overall, a person may have a 25% chance of

surviving an arrest that occurs right in front of somebody else. However, survival chances are a lot less for the subcategory of persons who present asystolic and gets a pulses back 15 minutes out, or those over 70 years old. There is still a chance of survival; it's just less. Prediction is a multifactorial equation. As a result, a physician's judgment is probably required to predict futility. But even the best physicians have to be supplied all those predictive data points in some kind of context. I think what everyone wants is more data with which to make these decisions.

Rubenfeld: I couldn't agree with you more.



FACE TO FACE WITH CHANGE

AARC 41st Annual Convention and Exhibition
December 2-5 • Orlando, Florida

Research on ACLS Training—Which Methods Improve Skill & Knowledge Retention?

William Kaye MD

- I. Introduction
- II. History of Resuscitation Training
- II. Evaluation of Resuscitation Knowledge & Skills
 - A. Basic Resuscitation Skills
 - B. Advanced Resuscitation Skills
- V. Effect of Training on Survival from Cardiac Arrest
 - A. Prehospital Arrest
 - B. In-Hospital Arrest
- V. Advanced Resuscitation Training
- VI. Improving Retention & Performance
- VII. Improving Outcome following In-Hospital Cardiac Arrest
- VIII. Automated External Defibrillation by In-Hospital First Responders

Introduction

Long-term survival from cardiac arrest is actually determined by several related factors: the pre-existing condition of the patient, the cause of the arrest, the duration of the arrest before resuscitation is begun, basic and advanced cardiac life support (BLS and ACLS), postresuscitation care, and rehabilitation.¹ The effect of BLS and ACLS on long-term survival depends on both the response time and the quality of the intervention.

The sooner resuscitation is initiated after cardiac arrest, the more likely the patient is to survive. Eisenberg's group^{2,3} has unequivocally demonstrated that time to initiation of cardiopulmonary resuscitation (CPR) and time to

definitive care are the critical components associated with successful outcome. A short time to initiation of CPR without a relatively rapid time to definitive care is little better than if the initiation of CPR is delayed. Similarly, relatively immediate definitive care that follows a delay in the initiation of CPR also produces a poor outcome.

Both outside and within the hospital, there must exist an integrated system that includes mechanisms for communication and rapid response by properly trained and equipped personnel.⁴⁻⁷ Within hospital, all personnel who have direct patient contact must be trained in CPR and early defibrillation as first responders. All house officers as well as all medical and paramedical personnel who staff emergency, intensive and coronary care units, operating rooms and post-anesthesia recovery rooms, angiography and cardiac catheterization laboratories; and/or who are members of the hospital ACLS/Code team who respond to any arrest in the hospital must be trained in advanced resuscitation techniques. Each hospital must have a formalized plan for management of cardiac arrest.

History of Resuscitation Training

Following the publication by Kouwenhoven, Jude, and Knickerbocker of the landmark paper describing CPR,⁸ a curriculum for training was developed in 1961.^{9,10} The Resusci-Anne mannequin, designed by Åsmund Laerdal in the late 1950s to train school children in Norway in rescue

Dr Kaye is associated with the Division of Critical Care Medicine, Departments of Surgery and Medicine, Brown University—The Miriam Hospital, Providence, Rhode Island.

Parts of this paper are adapted from Kaye W, Mancini ME, Rallis SF, Mandel LP. Educational aspects: resuscitation training and evaluation. In: Cardiopulmonary resuscitation, Kaye W, Bircher NG (eds). New York: Churchill Livingstone 1989:177-222.

A version of this paper was presented by Dr Kaye at the RESPIRATORY CARE Journal Conference, "Resuscitation training and evaluation," October 21-23, 1994, Cancun, Mexico.

Reprints: William Kaye MD, The Miriam Hospital, Room 223, 164 Summit Avenue, Providence RI 02906.

breathing¹¹⁻¹³ was modified to permit external chest compression. Subsequently, through the Maryland Affiliate of the American Heart Association (AHA), the training program was made available to all AHA affiliates and its use rapidly spread throughout hospitals in the U.S.¹⁴⁻¹⁶

In an editorial in *Circulation* in 1962,¹⁷ closed-chest CPR was endorsed as a *medical* procedure. Subsequently, the method was reclassified as an *emergency* procedure in 1965¹⁸ and was endorsed by the AHA, American National Red Cross, and other national organizations. It was strongly recommended that the technique be applied by properly trained individuals in medical, dental, nursing, and allied health professions, and on rescue squads. At a conference on CPR sponsored by the National Research Council in 1966, the Ad Hoc Committee on Cardiopulmonary Resuscitation formulated guidelines both for the steps and sequences of exhaled air ventilation (rescue breathing) and CPR, and for training programs.¹⁴

During the 1960s, the feasibility of training the general public by lay instructors became established. At the National Conference on Standards for Cardiopulmonary Resuscitation and Emergency Cardiac Care (ECC) held in 1973,^{19,20} it was recommended that BLS CPR training programs be extended to the general public, and that integrated communitywide stratified programs of emergency cardiac care be generated as part of a comprehensive emergency medical services (EMS) system. The conference also recommended that training in CPR and emergency cardiac care be according to AHA Standards and that certification of competency at the various levels of life support be based on nationally standardized curricula that include both written and performance tests. These recommendations were reinforced at the 1979, 1985, and 1992 AHA Conferences of CPR and ECC^{4,21-23} and in the AHA BLS Training Manuals.²⁴⁻²⁹

Based on the 1973 Standards, in 1975 the AHA ACLS Training Program,^{30,31} modeled after a program developed by Carveth et al³² was introduced; instructors were trained; and the course was given to physicians and nurses, paramedics and paramedic students, and later to medical students throughout the U.S. The ACLS Textbook and Instructor's Manuals have been revised after each of the AHA Standards Conferences of 1979, 1985 and 1992, with the most recent revision in 1994.^{21,22,33-38}

Evaluation of Resuscitation Knowledge & Skills

The purpose of resuscitation education is to translate the science of BLS and ACLS into classroom performance with transfer to actual clinical practice. The goal of the AHA BLS and ACLS training programs is to provide cognitive, psychomotor, and interactive skills for physicians, nurses, and other health-care professionals (and for BLS,

lay citizens as well).^{4,29,37,38} These programs enable health care professionals to understand and respond appropriately in accordance with the AHA standards for CPR and emergency care, and maximize the likelihood of successful resuscitation.

Basic Resuscitation Skills

We recently reviewed 38 studies (published between 1966 and 1987) of the effect of course design and length, and refresher training on initial learning and retention of 1-rescuer CPR skills of physicians, nurses, emergency medical technicians at all levels, and the general public.³⁹ All studies³⁹⁻⁷² described poor retention of CPR skills by all groups studied, as early as 2 weeks after initial training. However, certain patterns emerged that are important not only for initial training but also for refresher training. Videotape or film viewing (even without mannequin practice), posters, instruction over the telephone, and self-instructional programs with mannequin practice using charts and interactive computer-video disk systems can provide some (if not all) CPR skills. Retention can be enhanced by video and film viewing, posters, written materials, and mannequin practice.^{44,45,62,63,65,68,69,71-76} Moreover, we have demonstrated that the problem of poor retention may be due to inadequate instruction and practice time, so that the student never learned in the first place!³⁹

Advanced Resuscitation Skills

ACLS skill-retention studies have also been discouraging. Lowenstein et al⁷⁷ found that ventricular tachycardia, ventricular fibrillation, asystole, and complete heart block were managed poorly by medical and surgical house officers, and prior ACLS training had no effect. Stross⁷⁸ found that physicians tested 1 year after ACLS training performed poorly in management of simulated rhythm disturbances, but retention was improved in those who had received quarterly mailings of reprints related to ACLS objectives and patient treatment problems.

Shannon et al⁷⁹ also tested 59 surgeons in airway management skills, use of monitor and defibrillator, and recognition and treatment of ventricular tachycardia and fibrillation, asystole, and complete heart block. Of those 51 surgeons, 51% were able to manage the airway and insert an endotracheal tube, 64% could operate the defibrillator, and 56% recognized all 4 malignant dysrhythmias, but only 19% treated the dysrhythmias correctly. Only defibrillation and dysrhythmia recognition were performed significantly better by those previously trained in ACLS.

Mancini and I⁸⁰ evaluated the performance of ACLS-trained medical residents as team leaders during actual resuscitations. Our evaluation identified improper CPR per-

formance, poor team leadership and patient assessment, difficulties with equipment, and failure to follow the correct treatment algorithms. We⁸¹ also tested, in the classroom without prior warning,³² medical residents and 9 critical care nurses previously trained in ACLS. Dysrhythmias were well recognized by all. The poorest performance was in assessment and problem solving. The problems that were identified in the classroom during Megacode testing were the same as those identified in the clinical setting.

Nelson⁸² studied the performance of 64 medical students who had been trained to intubate as part of an ACLS course, 2 to 3 months after training. The rate of successful intubation (confirmed by visualization) was only 70%. The most frequent errors were failure to check the light before intubation, use of the teeth as a fulcrum, and failure to check the cuff on the endotracheal tube.

Using a computer simulation program, Schwid and O'Donnell⁸³ tested anesthesiologists in the management of a cardiac arrest due to ventricular fibrillation in the operating room. Only 40% of anesthesia residents, 30% of attending anesthesiologists, and 20% of anesthesiologists in private practice managed the arrest correctly. The most common errors involved incorrect drug dosages (43%), incorrect defibrillator use, which included administering inadequate power levels and not defibrillating as often as recommended (40%), failure to hyperventilate (30%), and failure to turn off the anesthesia vaporizer (23%). Moreover, subjects with ACLS training in the prior 6 months did well (70% correct performance) whereas those with ACLS training more than 6 months prior to evaluation had markedly worse management. No anesthesiologist with ACLS training completed more than 2 years prior to evaluation and no anesthesiologist without previous ACLS training followed the recommended AHA algorithm.

Effect of Training on Survival from Cardiac Arrest

Prehospital Arrest

Studies of prehospital resuscitation from cardiac arrest due to ventricular fibrillation demonstrate strong support for the "Chain of Survival" concept: early activation of EMS system, prompt CPR, early defibrillation, and ACLS.⁸⁴⁻⁹⁸ Survival to hospital discharge following prehospital arrest in communities with a strong chain may exceed 30%.⁹⁸ There are no before-training-after-training studies regarding survival from cardiac arrest outside of the hospital. Nevertheless, it is clear that the beneficial effect on survival of bystander CPR and prompt defibrillation were dependent on the lay public trained in CPR and on emergency medical technicians (EMTs) and paramedics trained in early defibrillation and ACLS.¹⁰⁰

Moreover, without trained responders, survival can be predicted to be low, if not zero.

In-Hospital Arrests

Two reviews of 14,765 in-hospital resuscitations between 1960 and 1993^{99,100} demonstrated that average survival to hospital discharge has remained around 15%. No improvement has occurred during the 3 decades despite increased attention to resuscitation practice during that interval, including ACLS training that began in the U.S. in 1975. The Resuscitation Council of the United Kingdom (UK) recently studied 3,765 in-hospital arrests; although no organized ACLS program was in place, survival was also 15%.¹⁰¹ A few studies have looked specifically at the effects of training on survival from cardiac arrest in the hospital.

In 1979, Chamberlain's group¹⁰² demonstrated that ward nurses could be trained to recognize and manage ventricular fibrillation with defibrillation before arrival of either the arrest team or physicians. Such a program resulted in restoration of spontaneous circulation in 74% of patients who arrested because of ventricular fibrillation on the general medical wards, with 45% surviving to hospital discharge. However, no data regarding survival before implementation of this program were presented.

In 1986, Lowenstein et al.¹⁰³ using retrospective chart review, reported on the benefits of training physicians with the AHA ACLS Training Program. In the 6-month interval between September 1979 and March 1980, during which time there was no basic or advanced resuscitation training for house officers, short-term survival (restoration of pulse and blood pressure for at least 1 hour after cessation of resuscitation efforts) was 32% and long-term survival to hospital discharge was 13%. Following training of all house staff in ACLS, during the 6-month interval September 1982 to March 1983 short-term survival increased to 60% short term survival ($p = 0.009$), with 23% long-term survival. Unfortunately, the increase in long-term survival was not significantly different compared to pre-training.

Levy et al.¹⁰⁴ in 1992 in the UK investigated the effect of the Guidelines first published in 1984 by the Resuscitation Council of the UK on drug usage and survival to hospital discharge following in-hospital cardiac arrest. No formal ACLS training program existed during the period studied. Between 1982 and 1991 there were 667 arrests; yearly survival remained at 15%. Bicarbonate usage declined to zero by 1990, and epinephrine usage for asystole increased from 63 to 72% between 1982-1988 and to 92% by 1992. In short, although drug usage followed the UK Guidelines, survival to hospital discharge was unchanged.

Seidlin and Bridges¹⁰⁵ studied the effect on outcome of cardiac arrest following training of UK junior house offi-

cers in a 6-hour course. Initial and long-term survival were no different before and after training. They concluded that training of junior house officers alone is not an effective strategy for improving survival from cardiac arrest.

Saunders et al¹⁰⁶ evaluated the effect of ACLS training in a small rural hospital on long-term outcome following both prehospital and in-hospital cardiac arrest. In the 13-month period before ACLS training (no physician and 15% of nurses were "certified" in ACLS) there were 29 arrests; 2 patients (7%) had restoration of spontaneous circulation (ROSC) to allow transfer to an urban medical center, with 1 patient (3%) surviving to hospital discharge. In a second 13-month period following ACLS training (70% of nurses and physician assistants and 50% of physicians) there were 35 arrests. Of the 35 patients, 7 (20%) had ROSC and were transferred; 2 patients (7%) survived to hospital discharge. The difference was not significant. However, 85% of the arrests seen during the 2 study periods were prehospital, and EMTs provided only CPR with no defibrillation.

Improving Retention & Performance

It is important to discuss not only the reasons for retention, but also initial learning as well. Does the problem lie with the student, the curriculum, or the instructor?^{11,39,107-115} Two areas may relate to the student. A natural decrement occurs without reinforcement of newly acquired psychomotor skills as time-since-training increases. This may be improved both by initial overtraining and by periodic refresher training. And, as demonstrated by several of the studies of BLS, the student may never have learned the necessary skills in the first place.³⁸ The instructor may not allow adequate practice and remediation for skill (and knowledge) mastery; with ongoing evaluation to document student problems may be deficient.^{110,111,114,116}

There may be problems with the curriculum. Especially with basic CPR performance, the minimum number of ventilations and compressions, rate and ratio required to sustain life have never been defined as a basis for the Standards.^{8,9,14-28} Nevertheless, the basic CPR routine was specified down to the second in the previous AHA BLS Training Programs, with a specific number of required compressions and ventilations per minute, and with each cycle length, compression rate and depth, ventilation rate, and volume defined within a very narrow range. Many of these rigid criteria were based not on animal or human data, but on either performance by "experienced" CPR providers on recording CPR mannequins or by Committee consensus. The new BLS training program has addressed these issues, and performance criteria have become much less rigid.^{4,29}

The goals of the training programs and the minimum knowledge and skill objectives based on the goals must be

defined in order to determine course content. The length of the training program must be based on content, and the program must not include more information than necessary. Although performance objectives have been written and incorporated into checklists for teaching and testing,^{29,37} more information than necessary is being transmitted to the student in both the BLS and ACLS training programs.^{29,37} Patterns that are complex or that offer the learner many choices for action require more time to learn than may be available in the training programs. The individual skills and information must fit together and make sense before the learner can store the material in memory for later retrieval.¹⁰⁷⁻¹¹⁵ That an individual can learn immediately and effectively to perform if directions are simple and clear is demonstrated by the development and implementation of emergency CPR instruction by telephone.⁶⁸

Knowledge and skill testing based on the program objectives are important means of ensuring learning.¹¹⁶⁻¹¹⁸ Pretesting indicates to the student what is expected and establishes his or her level before training. Posttesting at the end of the course (or each session) not only allows evaluation of learning and the effectiveness of the course, by both student and teacher, but also reinforces learning. Retention testing, while also reinforcing learning, indicates whether refresher training is needed.¹¹⁸

Advanced Resuscitation Training

Junior house staff, junior nurses, respiratory therapists, and medical students usually transfer leadership for management of a cardiac arrest to a more senior person once he or she arrives. These junior personnel need only the knowledge and skills to manage effectively the first 5-10 minutes of cardiac arrest and to work as team members rather than as team leader once more-senior personnel arrive. The full 2-day AHA ACLS course may be too elaborate for beginners.

Based on this goal, we designed and validated two core ACLS Courses: a 6-h course in the UK taught in three 2-h sessions for junior house officers¹¹⁹ and a 10-12 h course in the U.S. taught in 2 or 3 sessions for junior house officers, critical care nurses, and medical students.¹²⁰ The courses emphasized practical skills, with most teaching in skill stations rather than by lectures. The core objectives included algorithms for life-threatening dysrhythmias; skills of CPR (airway and ventilation, intubation, monitoring and defibrillation, and assembly and operation of equipment); immediate postresuscitation care; principles and priorities of leader and team member roles (Megacode). Testing, followed AHA Guidelines and included written multiple-choice questions and cardiac-rhythm-recognition and treatment tests; and practical tests of bag-mask ventilation, intubation, and management of ventricular fibrillation. All subjects in the UK and U.S. were pretested (using the same

instruments), were taught the course, and were tested immediately following training and again 5-8 months later. All showed significant learning and retention. In the UK we demonstrated that experience as a junior house officer is not a substitute for practical training in advanced resuscitation. In the U.S. study, retention at 6 months by subjects who had been taught the core course was the same as those taught the AHA ACLS Course, but retention at 10 months by subjects taught the core ACLS Course was better. The core ACLS Course was shorter and as effective as the AHA ACLS Course. This core course served as a model for the new case-based 1-day ACLS course recently included in the new ACLS Instructors Manual.³⁷

A brief refresher course offered during the first year after training should improve retention. One year following this core course personnel should be offered a more advanced course. The advanced course would reinforce all knowledge and skills of the initial course, but would place more emphasis on cognitive knowledge, problem solving, management of the arrest beyond the first 5-10 minutes, including postresuscitation management, and leadership skills.

Rather than trying to design an ideal course, efforts should be directed to ensuring that performance standards and curriculum are based on the minimum required to maintain or save life (especially important for basic CPR and core ACLS); and that instructors teach in a standardized manner, allow adequate time for practice of psychomotor skills, and evaluate performance accurately. In addition, innovative means (described above in the studies on retention and performance) for refresher training need to be developed (ie, videos, films, posters, computer simulations, and written materials^{44,45,63,65,68,69,71-76}).

The new AHA ACLS Program has been designed to address some of these issues.³⁷ Subject-based knowledge and skills are no longer covered in individual lectures and teaching stations but are distributed in a series of case-based clinical scenarios that follow the ACLS Algorithms. Several different course designs and lengths are offered as examples and can be selected or modified by the course director, depending upon the needs of the course participants. Each course is designed to provide a positive learning environment to help participants master knowledge and skills, with evaluation used as a personal teaching tool rather than as a certification tool or to define "pass or fail." Studies evaluating the effect of this new paradigm on initial training and retention are not yet available.

Improving Outcome following In-Hospital Cardiac Arrest

As mentioned above, survival to hospital discharge following prehospital arrest in communities with a strong Chain of Survival may exceed 30%. But, long-term sur-

vival following in-hospital cardiac arrest remains much lower, averaging 15%. With immediate care available, should not in-hospital survival be greater than prehospital?

Most prehospital arrests are due to primary ventricular fibrillation from myocardial ischemia in patients who appear otherwise healthy.^{2,3,84-86,121} In the coronary care unit, survival following ventricular fibrillation arrest may approach 90%.¹²²⁻¹²⁵ But in-hospital arrest outside of CCU usually occurs in sick patients with single or multiple organ system dysfunction. Moreover, despite better acceptance of policies for withholding CPR, the federal law mandating advanced directives, and a growing body of data that allow prediction of poor outcome following arrest,¹²⁶⁻¹²⁹ many patients still receive inappropriate CPR.

Automated External Defibrillation by In-Hospital First Responders

Better BLS and ACLS alone may not be the real solution.¹ The AHA has expanded early defibrillation as the standard of care^{4,84,96,130} to include the use of automated external defibrillators (AEDs) by first responders trained in BLS, for both prehospital and in-hospital cardiac arrest due to pulseless ventricular fibrillation.¹³⁰⁻¹⁴¹

The rationale for early defibrillation is;^{84,96,121,130,139-141}

- The most frequent initial rhythm in sudden cardiac arrest is ventricular fibrillation.
- Ventricular fibrillation is chaotic electrical activity of the heart with no pumping action.
- The most effective treatment for ventricular fibrillation is electrical defibrillation.
- The probability of successful defibrillation diminishes rapidly over time.
- Ventricular fibrillation tends to deteriorate to asystole within a few minutes.

Early defibrillation using AEDs by first responders is in place in several prehospital emergency medical systems.⁸⁸⁻⁹⁸ But, no in-hospital first-responder defibrillation programs exist except in critical care areas where nurses are trained to recognize and treat ventricular fibrillation with conventional defibrillators. Outside critical care areas, staff nurses trained in BLS (usually the first responders) provide only CPR until the arrival of the resuscitation team. The resuscitation team provides defibrillation and advanced life support.

The Chain of Survival should operate both out of hospital and in hospital. Because survival is inversely proportional to the duration of ventricular fibrillation, AEDs used by first responders (BLS-AED) in-hospital should shorten the time to defibrillation and thereby increase survival to hospital discharge of hospitalized patients with pulseless ventricular fibrillation.

We have recently completed a study to determine, before implementing an in-hospital first responder AED program, whether staff outside critical care areas who were already proficient in BLS could be easily trained to use AEDs and would retain these skills.¹⁴² Participants were 140 nurses who had previously learned BLS and were the staff from three medical/surgical nursing units in two university teaching hospitals. The nurses were taught how to use the Heartstart 1000s, (Ladler Medical Corp, Armonk NY) a light-weight portable shock-advisory AED, in a 2-hour class with an instructor-and-mannequin-to-student ratio of 1:5. The course emphasized hands-on practice of the BLS-AED algorithm on a computerized mannequin. Using a similar scenario, each nurse was evaluated on the computerized mannequin immediately after training (post-test). At 1-3, 4-6, and 7-9 months after initial training, convenience samples of the cohort in three different groups were evaluated for retention. Satisfactory performance was defined as delivery of the first AED shock within 2 minutes of recognition of the arrest. On the post-test after training, 139/140 nurses (=99%) demonstrated satisfactory performance. For retention, 77 nurses were evaluated; 31/32 between 1-3 months, 18/18 between 4-6 months, and 24/27 between 7-9 months after initial training (95% overall) performed satisfactorily.

Automated external defibrillation is a *basic-life support* skill and should be incorporated into *all* BLS training programs (BLS-AED) for *all* hospital personnel expected to respond to a cardiac arrest (including physicians, nurses, physician assistants, respiratory therapists, radiology technicians, and students), with rapid defibrillation taking priority over CPR.¹⁴²⁻¹⁴⁴ Because the dysrhythmia detection algorithms in the AEDs are sensitive and specific in recognizing shockable rhythms,¹⁴⁵ there is no need to train the operator to recognize ventricular fibrillation or ventricular tachycardia or, for that matter, any dysrhythmia. The first responders need only recognize that the patient is unresponsive, apneic, and pulseless before attaching and activating the AED. Less training time is needed to learn to use an AED than to learn dysrhythmia recognition and operation of a conventional defibrillator. Moreover, as our retention data confirm application of an easy-to-operate AED remains effective in an environment in which first responders may have only rare exposure to a cardiac arrest.

Identifying those patients who should not be offered CPR, terminating resuscitation attempts when outcome can be predicted to be poor, early first-responder-defibrillation, effective CPR if defibrillation is unsuccessful or not indicated, and early ACLS by well-trained providers should allow long-term survival from in-hospital arrest to approach that of prehospital arrest.¹⁴⁶

REFERENCES

1. Kaye W, Mancini ME, Rallis SF, et al. Can better basic and advanced cardiac life support improve outcome from cardiac arrest? *Crit Care Med* 1985;13:916.
2. Eisenberg M, Bergner L, Hallstrom A. Paramedic programs and out-of-hospital cardiac arrest: I. Factors associated with successful resuscitation. *Am J Public Health* 1979;69:30.
3. Eisenberg MS, Bergner L, Hallstrom A. Cardiac resuscitation in the community. *JAMA* 1979;241:1905.
4. American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiac care. *JAMA* 1992;268:2171-2302.
5. Evans TR, editor. ABC of resuscitation. London, Br Med J, 1986.
6. Royal College of Physicians. Resuscitation from cardiopulmonary arrest. Training and organization. A Report of the Royal College of Physicians. *J R Coll Physicians Lond*. 1987;21:1.
7. American Heart Association. Putting it all together. In: *Textbook of advanced cardiac life support*. Dallas: American Heart Association 1987: 235-248.
8. Kouwenhoven WB, Jude JR, Knickerbocker GG. Closed-chest cardiac massage. *JAMA* 1960;173:1064.
9. Safar P, Brown TC, Holty WH, et al. Ventilation and circulation with closed chest cardiac massage in man. *JAMA* 1961;176:574.
10. Jude JR, Elam JO. Fundamentals of cardiopulmonary resuscitation. Philadelphia: FA Davis, 1965.
11. Elam JO, Ruben HM, Bittner TJ. Training laymen in emergency resuscitation. *Anesth Analg* 1961;40:603.
12. Lind B. Teaching mouth-to-mouth resuscitation in primary schools. *Acta Anaesth Scand (suppl)* 1961;9:63.
13. Lind B, Stovner J. Mouth-to-mouth resuscitation in Norway. *JAMA* 1963;185:933.
14. Cardiopulmonary Resuscitation Conference Proceedings. Washington, D.C. National Research Council, 1966.
15. American Heart Association. Standards for cardiopulmonary resuscitation and emergency cardiac care. *JAMA* 1966;198:3731.
16. American Heart Association. Cardiopulmonary resuscitation. A manual for instructors. New York: American Heart Association, 1971.
17. The closed chest method of cardiopulmonary resuscitation. Benefits and hazards (editorial). *Circulation* 1962;26:324.
18. The closed-chest method of cardiopulmonary resuscitation—Revised statement (editorial). *Circulation* 1965;31:641.
19. American Heart Association. Standards for cardiopulmonary resuscitation (CPR) and emergency cardiac care (ECC). *JAMA* 1974;227(suppl):796,797,833-868.
20. American Heart Association. Proceedings of the National Conference on Standards for Cardiopulmonary Resuscitation (CPR) and Emergency Cardiac Care (ECC) Dallas: American Heart Association, 1975.
21. American Heart Association. Standards and guidelines for cardiopulmonary resuscitation (CPR) and emergency cardiac care (ECC). *JAMA* 1980;244:453.
22. American Heart Association. Standards and guidelines for cardiopulmonary resuscitation (CPR) and emergency cardiac care (ECC). *JAMA* 1986;255:2841.
23. Proceedings of the 1985 National Conference on Standards and Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiac Care. *Circulation* 1986;74 (suppl IV):1-153.
24. American Heart Association. A manual for instructors of basic cardiac life support. Dallas: American Heart Association, 1977.
25. American Heart Association. A manual for instructors of basic cardiac life support. Dallas: American Heart Association, 1980, 1981.
26. American Heart Association. Instructors manual for basic life support. Dallas: American Heart Association, 1987.

27. American Heart Association. Heartsaver manual. Dallas: American Heart Association, 1987.
28. American Heart Association. Healthcare provider's manual for basic life support. Dallas: American Heart Association, 1988.
29. American Heart Association. Basic life support instructor's manual. Dallas: American Heart Association, 1994.
30. American Heart Association. Advanced cardiac life support. Dallas: American Heart Association, 1975.
31. American Heart Association. A manual for instructors of advanced cardiac life support. Dallas: American Heart Association, 1977.
32. Carveth SW, Burnap TK, Bechtel J, McIntyre K, Donegan J, Buchman RJ, Reese HE. Training in advanced cardiac life support. *JAMA* 1976;235:2311.
33. MacIntyre KM, Luis AAJ (editors). Textbook of advanced cardiac life support. Dallas: American Heart Association, 1981.
34. Kaye W, Paraskos JA, editors. Instructors manual for advanced cardiac life support. Dallas, American Heart Association, 1982.
35. American Heart Association. Textbook of advanced cardiac life support. Dallas: American Heart Association, 1987.
36. American Heart Association. Instructors manual for advanced cardiac life support. Dallas: American Heart Association, 1988.
37. Billi DIE, Cummins OR, editors. Advanced cardiac life support instructor's manual. Dallas: American Heart Association, 1994.
38. Cummins OR (editor.). Textbook of advanced cardiac life support. Dallas: American Heart Association, 1994.
39. Kaye W, Rallis FS, Mancini ME, Linhares KC, Angell ML, Donovan DS, et al. The problem of poor retention of cardiopulmonary resuscitation skills may lie with the instructor, not the learner or the curriculum. *Resuscitation* 1991;21:67-87.
40. Curry L, Gass D. Effects of training in cardiopulmonary resuscitation on competence and patient outcome. *Can Med Assoc J* 1987;137:491.
41. Thrasher CL. Physician one-rescuer CPR performance during advanced cardiac life support provider instruction. *Crit Care Med* 1987;15:181.
42. Kaye W, Mancini ME. Retention of cardiopulmonary resuscitation skills by physicians, nurses and general public. *Crit Care Med*. 1986;14:620.
43. Mancini ME, Kaye W. The effect of time since training on house officers' retention of cardiopulmonary resuscitation skills. *Am J Emerg Med* 1985; 3:31.
44. Winchell S, Safar P. Teaching and testing lay and paramedical personnel in cardiopulmonary resuscitation. *Anesth Analg* 1966;45:441.
45. Berkebile P, Berson D, Ersoz C, et al. Public education in heart-lung resuscitation. Evaluation of three self-training methods in teenagers. In: Proceedings of National Conference on Standards for Cardiopulmonary Resuscitation (CPR) and Emergency Cardiac Care (ECC) Dallas, Texas American Heart Association. 1975:13-23.
46. Vanderschmidt H, Burnap T, Thwaites JK. Evaluation of a cardio-pulmonary resuscitation course for secondary schools. *Med Care*. 1975;13:763.
47. Vanderschmidt H, Burnap T, Thwaites JK. Evaluation of cardiopulmonary resuscitation course for secondary schools retention study. *Med Care* 1976;14:181.
48. Ramirez AG, Weaver FJ, Raizner AE, et al. The efficacy of lay CPR instruction: an evaluation. *Am J Public Health*. 1977;67:1093.
49. Webb DD, Lambrew C. Evaluation of physician skills in cardiopulmonary resuscitation. *J Am Coll. Emergency Physicians* 1978;7:387.
50. Kortilla K, Vertio H, Savolainen K. Importance of using proper techniques to teach cardiopulmonary resuscitation to laymen. *Acta Anaesthesiol Scand* 1979;23:235.
51. Weaver FJ, Ramirez AG, Dorfman SB, et al. Trainees' retention of cardiopulmonary resuscitation—How quickly they forget. *JAMA* 1979;241:901.
52. Tweed WA, Wilson E, Isfeld B. Retention of cardiopulmonary resuscitation skills after initial overtraining. *Crit Care Med* 1980;8:651.
53. Deliere HM, Schneider LE. A study of cardiopulmonary resuscitation technical skill retention among trained EMT-As. *Emerg Med Tech J* 1980;4:57.
54. Latman NS, Wooley K. Knowledge and skill retention of emergency care attendants, EMT-As, and EMT-Ps. *Ann Emerg Med* 1980;9:183.
55. Grogono AW, Johnson MM, Jastremski MS, Russell RF. Educational graffiti: better use of the lavatory wall. *Lancet* 1982;i:1175.
56. Mandel LP, Cobb LA. Initial and long-term competency of citizens trained in CPR. *Emerg Health Services Q* 1982;1:49.
57. Gombeski WR, Effron DM, Ramirez AG, et al. Impact on retention: comparison of two CPR training programs. *Public Health Briefs* 1982;72:849.
58. Gass DA, Curry L. Physicians' and nurses' retention of knowledge and skill after training in cardiopulmonary resuscitation. *Can Med Assoc J* 1983; 128:550.
59. Wilson E, Brooks B, Tweed WA. CPR skills retention of lay basic rescuers. *Ann Emerg Med* 1983;12:482.
60. Martin WJ, Loomis JH, Lloyd CW. CPR skills: achievement and retention under stringent and relaxed criteria. *Am J Public Health* 1983; 73:1310.
61. Fossel M, Kiskaddon RT, Sternbach GL. Retention of cardiopulmonary resuscitation skills by medical students. *J Med Educ* 1983;58:568.
62. Kaye W, Montgomery W, Hon D, et al. Interactive computer-videodisc CPR training and testing. *Circulation* 1983; 68(suppl III):III-14.
63. Lane JC, Nagase Y. Feasibility of teaching resuscitation by television in Brazil. *Disaster Medicine* 1983;1:89.
64. Donovan DS, Zajano NC, Finger JA Jr, et al. Five minutes to live: a study of the effectiveness of three hours training in one-person CPR. Center for Evaluation and Research, Rhode Island College, 1983.
65. Bircher N, Safar P. Life supporting first aid (LSFA) and infant CPR (ICPR) self-training in children. *Crit Care Med* 1983;11:251.
66. Nelson M, Brown CG. CPR instruction modular versus lecture course. *Ann Emerg Med* 1984;13:118.
67. Friesen L, Stotts NA. Retention of basic cardiac life support content: the effect of two teaching methods. *J Nurs Ed* 1984;23:184.
68. Carter WB, Eisenberg MS, Hallstrom AP, et al. Development and implementation of emergency CPR instructions via telephone. *Ann Emerg Med* 1984;13(Part 1):695.
69. Edwards MJA, Hannah KJ. An examination of the use of interactive videodisc cardiopulmonary resuscitation instruction for the lay community. *Comput Nurs* 1985;3:250.
70. Van Kalmthout PM, Speth PAJ, Rutten JR, et al. Evaluation of lay skills in cardiopulmonary resuscitation. *Br Heart J* 1985;53:562.
71. Mandel LP, Cobb LA. Reinforcing CPR skills without mannequin practice. *Ann Emerg Med* 1987;16:1117.
72. Schlager J, Hayes JG, Turino GM, et al. The effectiveness of film and videotape in teaching cardiopulmonary resuscitation to the lay public. *NY State J Med* 1987;87:382.
73. Berkebile P. Education in cardiopulmonary-cerebral resuscitation (CPCR) using self-training methods. *Disaster Med* 1983;1:75.
74. Safar P, Berkebile PE, Scott MA, et al. Education research on life-supporting first aid (LSFA) and CPR self-training systems (STS). *Crit Care Med* 1981;9:403.

75. Scott M, Safar P, Berkebile P, et al. Cardiopulmonary resuscitation (CPR) basic and advanced (BLS, ALS) self-training systems (STS) for paramedical and medical personnel. *Disaster Medicine* 1983;1:96.
76. Uhley HN, Fox C, Levy E, et al. Enhancement of recall of cardiopulmonary resuscitation (CPR): use of a moving-image card to review the dynamics of CPR. *Emerg Lung* 1979;8:517.
77. Lowenstein S, Libby L, Mountain R, et al. Cardiopulmonary resuscitation by medical and surgical house officers. *Lancet*. 1981;2:679.
78. Stross JK. Maintaining competency in advanced cardiac life support skills. *JAMA* 1983;249:3339.
79. Shannon FL, Jurkovich GJ, Hansbrough JF. Assessment of the proficiency of the surgeon in providing basic and advanced cardiac life support. *SG & O* 1984;159:9.
80. Mancini ME, Kaye W. A comparison of the results of Mega Code testing with ACLS performance during actual resuscitations—a pilot study. *Crit Care Med* 1987;15:368.
81. Kaye W, Mancini ME. Use of the Mega Code to evaluate team leader performance during advanced cardiac life support. *Crit Care Med* 1986;14:99.
82. Nelson MS. Medical student retention of intubation skills. *Ann Emerg Med* 1989;18:1059-1061.
83. Schwid HA, O'Donnell. Anesthesiologists' management of simulated critical incidents. *Anesthesiology* 1992;76:495-501.
84. Cummins RO, Ornato JP, Thies WH, Pepe PE, Ornato JP, Advanced Life Support Subcommittee and Emergency Cardiac Care Committee of the American Heart Association. Improving survival from sudden cardiac arrest: the "chain of survival" concept. *Circulation* 1991;83:1832-1847.
85. Weaver WD, Cobb LA, Hallstrom AP, Copass MK, Ray R, Emery M, Fahrenbruch C. Considerations for Improving survival from out-of-hospital cardiac arrest. *Ann Emerg Med* 1986;15:1181-1186.
86. Weaver WD, Cobb LA, Hallstrom AP, Fahrenbruch C, Copass MK, Ray R. Factors influencing survival after out-of-hospital cardiac arrest. *J Am Coll Cardiol* 1986;7:752-757.
87. Spaite DW, Hanlon T, Criss EA, Valenzuela TD, Wright AL, Keeley KT, Meislin HW. Prehospital cardiac arrest: the impact of witnessed collapse and bystander CPR in a metropolitan EMS system with short response times. *Ann Emerg Med* 1990;19:1264-1269.
88. Eisenberg MS, Hallstrom AP, Copass MK, Bergner L, Short F, Pierce J. Treatment of ventricular fibrillation: emergency medical technician defibrillation and paramedic services. *JAMA* 1984;251:1723-1726.
89. Stults KR, Brown DD, Schug VL, Bean JA. Prehospital defibrillation performed by emergency medical technicians in rural communities. *N Engl J Med* 1984;310:219-223.
90. Weaver WD, Copass MK, Bui D, Ray R, Hallstrom AP, Cobb LA. Improved neurologic recovery and survival after early defibrillation. *Circulation*, 1984;69:943-948.
91. White RD. EMT-defibrillation: time for controlled implementation of effective treatment. *Emerg Cardiac Care Newsletter* 1986;8:1-3.
92. Cummins RO. EMT-Defibrillation: national guidelines for implementation. *Am J Emerg Med* 1987;5:254-257.
93. Cummins RO, Eisenberg MS, Litwin PE, Graves JR, Hearne TR, Hallstrom AP. Automatic external defibrillators used by emergency medical technicians. *JAMA* 1987;257:1605-1610.
94. Paris PMP. EMT-defibrillation: a recipe for saving lives. *Am J Emerg Med* 1988;6:282-287.
95. Olson DW, LaRochelle J, Fark D, Arahamian C, Aufderheide TP, Mateur JR, et al. EMT-Defibrillation: the Wisconsin experience. *Ann Emerg Med* 1989;18:806-811.
96. Cummins RO. From concept to standard-of-care? review of the clinical experience with automated external defibrillators. *Ann Emerg Med* 1989;18:1269-1275.
97. Hargarten KM, Stueven HA, Waite EM, Olson DW, Mateur JR, Aufderheide TP, Darin JC. Prehospital experience with defibrillation of coarse ventricular fibrillation: a ten-year review. *Ann Emerg Med* 1990;19:157-162.
98. Eisenberg MS, Horwood BT, Cummins RO, Reynolds-Haertle R, Hearne TR. Cardiac arrest and resuscitation: a tale of 29 cities. *Ann Emerg Med* 1990;19:179-186.
99. McGrath RB. In-house cardiopulmonary resuscitation—after a quarter of a century. *Ann Emerg Med* 1987;16:1365-1368.
100. Jastremski MS. In-hospital cardiac arrest. *Ann Emerg Med* 1993;22:113-117.
101. Tunstall-Pedoe H, Bailey L, Chamberlain DA, Marsden AK, Ward ME, Zideman DA. Survey of 3765 cardiopulmonary resuscitations in British hospitals (the BRESUS study): methods and overall results. *Br Med J* 1992;304:1346-1351.
102. Mackintosh AF, Crabb ME, Brennan H, et al. Hospital resuscitation from ventricular fibrillation in Brighton. *Br Med J* 1979;511.
103. Lowenstein SR, Sabyan EM, Lassen CF, et al. Benefits of training physicians in advanced cardiac life support. *Chest* 1986;89:512.
104. Levy RD, Rhoden WE, Shearer K, Varley E, Brooks NH. An audit of drug usage for in-hospital cardiopulmonary resuscitation. *Eur Heart J* 1992;13:1665-1668.
105. Seidlen PH, Bridges AB. Cardiopulmonary resuscitation: effect of training junior house officers on outcome of cardiac arrest. *J R Coll Physicians Lond* 1993;27:52-53.
106. Saunders AB, Berg RA, Burrell M, Genova RT, Kern KB, Ewy GA. The efficacy of an ACLS training program for resuscitation from cardiac arrest in a rural community. *Ann Emerg Med* 1994;23:56-59.
107. Dewey J. How we think. New York: MacMillan Company, 1910.
108. Tyler R. Principles of curriculum and instruction. Chicago: University of Chicago Press, 1949.
109. Gudmundsen A. Teaching psychomotor skills. *J Nurs Ed* 1975;14:23.
110. Ammons RB, Farr RG, Bloch E, et al. Long-term retention of perceptual motor skills. *J Exp Psychol* 1958;55:318.
111. Melnick MJ. Effects of overlearning on the retention of a gross motor skill. *Res Q* 1971;42:60.
112. Corbin CB. Effects of mental practice on skill development after controlled practice. *Res Q* 1967;38:534.
113. Stebbins RJ. A comparison of the effects of physical and mental practice in learning a motor skill. *Res Q* 1968;39:714.
114. Robb M. Feedback and skill learning. *Res Q* 1968;39:175.
115. Barbe WB, Swassing RH, Milone MN. Teaching through modality strengths: concepts and practices. Columbus, Ohio: Zaner-Bloser, 1979.
116. Fitz-Gibbon CT, Morris LL. How to design a program evaluation. Beverly Hills: Sage Publications, 1978.
117. Mancini ME, Kaye W. Development and validation of an objective criterion-referenced tool for evaluation of advanced cardiac life support performance. *Heart Lung* 1985;14:299.
118. Kaye W, Mancini ME, Rallis SF. Advanced cardiac life support refresher course using objective-based Mega Code testing. *Crit Care Med* 1987;15:55.
119. Kaye W, Wynne G, Marteau T, Dubin HG, Rallis SF, Simons RS, Evans TR. An advanced resuscitation training course for preregistered house officers. *J R Coll Physicians Lond* 1990;24:51-54.
120. Kaye W, Dubin H, Rallis SF. A core advanced cardiac life support (ACLS) course for junior house officers (PG-1s). *Clin Res* 1988;36:371A.

121. Bayou de Luna A, Coumel P, Leclercq JF. Ambulatory sudden cardiac death: mechanisms of production of fatal arrhythmia on the basis of data from 157 cases. *Am Heart J* 1989;117:151-159.
122. Conley MJ, McNeer JF, Lee KL, Wagner GS, Rosati RA. Cardiac arrest complicating acute myocardial infarction: predictability and prognosis. *Am J Cardiol* 1977;39:7-12.
123. Carruth JE, Silverman ME. Ventricular fibrillation complicating acute myocardial infarction: reasons against the routine use of lidocaine. *Am Heart J* 1982;9:545-550.
124. Dubois C, Smeets JP, Demoulin JC, Piérard L, Goidart G, Henard L, et al. Incidence, clinical significance and prognosis of ventricular fibrillation in the early phase of myocardial infarction. *Eur Heart J* 1986;7:945-951.
125. Goldberg RJ, Gore JM, Haffajee CI, Alpert JS, Dalen JE. Outcome after cardiac arrest during acute myocardial infarction. *Am J Cardiol* 1987;59:251-255.
126. Bedell SE, Delbanco TL, Cook F, Epstein FH. Survival After Cardiopulmonary Resuscitation in the Hospital. *N Engl J Med* 1983;309:569-576.
127. Rosenbaum EA, Shenkman L. Predicting outcome of in-hospital cardiopulmonary resuscitation. *Crit Care Med* 1988;16:583-586.
128. Roberts D, Landolfo K, Light RB, Dobson K. Early predictors of mortality for hospitalized patients suffering cardiopulmonary arrest. *Chest* 1990;97:413-419.
129. Jensen GVH, Torp-Pedersen C, Kober L. Prognosis of ventricular fibrillation in hospital. *Eur Heart J* 1992;13:1185-1188.
130. Cummins RO, Thies W, Paraskos J, Kerber RE, Billi JE, Seidel J, et al. Encouraging early defibrillation: the American Heart Association and automated external defibrillators. *Ann Emerg Med* 1990;19:1245-1248.
131. Cummins RO, Eisenberg MS, Bergner L, Hallstrom AP, Hearne T, Murray JA. Automatic external defibrillation: evaluations of its role in the home and in emergency medical services. *Ann Emerg Med* 1984;13:789-801.
132. Cummins RO, Eisenberg MS, Moore JE, Hearne TR, Andersen E, Weendt R, et al. Automatic external defibrillators: clinical, training, psychological, and public health issues. *Ann Emerg Med* 1985;14:755-760.
133. Cummins RO, Eisenberg MS, Stults KR. Automatic external defibrillators: clinical issues for cardiology. *Circulation* 1986;73:381-385.
134. Stults KR, Brown DD, Kerber RE. Efficacy of an automated external defibrillator in the management of out-of-hospital cardiac arrest: validation of the diagnostic algorithm and initial experience in a rural environment. *Circulation* 1986;73:701-709.
135. Stults KR, Cummins RO. Fully automatic vs shock advisory defibrillators: what are the issues? *J Emerg Med Services* 1987;12:71-73.
136. Weaver WD, Hill D, Fahrenbruch CE, Copass MK, Martin JS, Cobb LA, Hallstrom AP. Use of the automatic external defibrillator in the management of out-of-hospital cardiac arrest. *N Engl J Med* 1988;319:661-666.
137. Bock JJ. Automatic external defibrillators. *Ann Emerg Med* 1989;18:1264-1268.
138. Edwards DG. Development of a decision algorithm for semiautomatic defibrillator. *Ann Emerg Med* 1989;18:1276-1279.
139. Cummins RO, Thies W. Automated external defibrillators and ACLS: a new initiative from the American Heart Association. *Am J Emerg Med* 1991;9:91-94.
140. American Heart Association. Automated external defibrillation. In: *Textbook of advanced cardiac life support*, Dallas: American Heart Association, 1990, Chapter 20.
141. American Heart Association. Training in use of automated external defibrillators. In: *Instructor manual for advanced cardiac life support*. Dallas: American Heart Association, 1990, Chapter 6 Supplement (Early Defibrillation).
142. Kaye W, Mancini JE, Giuliano KK, Richards N, Nagid DM, Marler CA, Sawyer-Silva S. Strengthening the in-hospital chain of survival with rapid defibrillation by first responders using automated external defibrillators: training and retention issues. *Ann Emerg Med* 1995;25:163-168.
143. DeBono D. Resuscitation—time for a re-think? *Q J Med* 1991;81:959-960.
144. Brenner BE, Kauffman J. Reluctance of internists and medical nurses to perform mouth-to-mouth resuscitation. *Arch Intern Med* 1993;153:1763-1769.
145. Cummins RO, Stults KR, Haggard B, Kerber RE, Schaeffer S, Brown DD. A new rhythm library for testing automatic external defibrillators: performance of three devices. *J Am Coll Cardiol* 1988;11:597-602.
146. Kaye W. Improving outcome from in-hospital cardiac arrest with a reorganized and strengthened in-hospital chain of survival. 1994, submitted for publication, 1995.

Kaye Discussion

Barnes: I found that really interesting, Bill. You said you're using the written exam differently now. Could you expand a little further under the new ACLS training program; how are you using the written exam?

Kaye: We have two written exams as posttests. One is multiple choice, the other arrhythmia recognition and treatment. If students do poorly, we bring them back in small groups and go over each question, review the correct answers, and have them take

the test again. Our job is to make them learn, not pass or fail. The CPR police have resigned.

Durbin: They still exist at the state level.

Bishop: Your slide referred to Schwid & O'Donnell's study. The authors also publish a CPR simulation. Their study is excellent. I'd like to hear your comment on simulation as an educational tool because simulated cases are a way of teaching and refreshing people on ACLS. I'd also like to point out that the high error rate you noted

among anesthesiologists in the study by Schwid included many trivial errors that wouldn't affect the patient outcome—such as whether you felt for a pulse after each shock, which, in fact, has been changed since the time of the study. Many of the errors would not affect outcome. I think it, probably, way underestimates the knowledge of providers.

1. Schwid HA, O'Donnell D. The anesthesia simulator-recorder: a device to train and evaluate anesthesiologists' responses to critical incidents. *Anesthesiology* 1990;72:191-197.

Kaye: That is probably true of all studies—one can say that. But, it does tell us that we need to improve our teaching methodology and retention programs. As a way of reviewing and reinforcing knowledge and skills—arrhythmia, algorithms, protocols, patient management issues—I think computer simulations may be very effective. Had these anesthesiologists been given a mannequin and a defibrillator with monitor, they may well have done better. I hear what you're saying, but I really don't have much experience with computer simulations, so I really can't comment. Maybe somebody else can.

Boudin: I'll be addressing interactive video disk technology in my presentation.

Mathews: I just want to comment on your concept of the first 5 minutes being the focus of the training. I have a problem with that because the majority of hospitals in the United States are not teaching hospitals and may not have house staff. If they don't have obstetrical services, they may not have an anesthesiologist, which means that the ER doc drops whatever he or she is doing and runs to the code. I suggest that to say that the only thing that should be taught is the first 5 minutes might be an error, if you are not within a university teaching hospital complex. In the general community, that might be disastrous, when a whole group of people are doing the first 5 minutes over and over again because it may take 15, 20, 30 minutes for a trained physician to show up.

Kaye: I hear what you're saying, and I tried to indicate that this was based on 2 hospitals, 1 in the UK and 1 in the U.S. with house staff. I mentioned that the paramedics—the prehospital responders—maybe have different requirements because there is no additional help, and, perhaps, in smaller hospitals without house staff that

may also be so. But I still believe very strongly that ACLS training should be done in short, 1-day or shorter segments, and that maybe segment Number 1 should be management of the first 5-10 minutes—the basics. Then, later on go on to the more elaborate maneuvers.

Mathews: I agree with that. In fact, our experience with our respiratory therapy students is that we run a 6-to-8 week course (2 hours/week) on ACLS. Then they also go through the regular ACLS Provider course. And they do very well.

Kaye: I think one tailors the program for the audience.

Mathews: So we continue reinforcement; we make use of techniques like the serial-position effect (ie, what is presented first and last is retained longer and more accurately than material presented in the midportion of the class) and keep on pushing all of this material so they become very competent at this. Some of our classes have done exceedingly well. They've done much better than physicians and nurses.

Kaye: But it's a resource issue, and I'm not sure that every institution, no matter what size, can afford a 6-month training program for the RCPs.

Mathews: You're right, you're absolutely right.

Kaye: I think each course must be designed to address individual needs; but all I'm saying is keep it simple. For example, lidocaine. Do you really need to know the complex kinetics? It's nice to know about the alpha and beta half-life. You need to know when to use it, what dose to give, and some of the caution words.

Mathews: And what outcome to expect.

Kaye: Defibrillation—I don't even care if you know what a square wave is versus a trapezoidal something else. But, how do you turn the machine on? How do you push the button? That's what people get hung up on. Where do you put the paddles? That's my message—keep it to the basics.

Sanders: I think one of the issues is data collection. Your study in Virginia is a good example of the importance of collecting data. Trauma centers are an example. In order to be a trauma center and keep your designation, you must keep a database of patient injuries, outcomes, and demographic information. This database can compare your outcomes to those in other hospitals and can be used for CQI. I think we should keep similar databases for cardiac-arrest patients to assess the quality of our arrest-response teams.

Kaye: I think that in-hospital resuscitation, within the Heart Association, has somewhat gotten lost. AHA has emphasized prehospital resuscitation, which is terribly important, but we also need to look inward to in-hospital. I would encourage AHA, to develop for in-hospital a standardized data instrument based on the prehospital Utstein model.¹

1. Cummins RO, Chamberlain DA, Abramson NS, et al. Recommended guidelines for uniform reporting of data from out-of-hospital cardiac arrest: the Utstein style. *Circulation* 1991; 84: 960-075.

Barnes: Bill, this touches on Paul's (Pepe), description of the team approach with the fire department, where people have designated responsibilities. Have you done much with simulated cardiac arrests and bringing people together, to see what kind of roles they took and if a leader emerged?

Kaye: We have tried that; after we developed the Megacode in the 70s in

Rhode Island, it became part of the national program in the early 80s. We identified the appropriate team members—a leader, an airway person, a chest-compressor person, an I.V. person (I.V. drugs), a defibrillation person, and we try to teach that. We don't have a designated code team, which makes it a little more difficult. We need to reinforce the idea that there are specific, basic roles that are required for resuscitation. I like Paul Pepe's ABCD—that's really neat. I'm going to copy that, if I may, for in-hospital.

Pepe: Please do, Bill. I believe it makes a difference.

Barnes: I think the problem is that we have interdisciplinary courses and training, and yet it may be impossible in hospitals. I think there is a distinct advantage if there can be a code team—a common set of people who train together, so, it isn't always just who shows up on that particular day.

Kaye: Our RCPs actually do have primary responsibility for noninvasive airway management, which concerns me because our residents don't take the opportunity to do bag-mask ventilation. The therapists are the AB people.

Pepe: The way we use it is that everybody (ie, every firefighter) is actually trained in the ABCDE thing—but on a given day the captain in charge that day says "OK, today you're (pointing to one man) going to be the first linesman, you're (pointing to another) going to be the plugman," and each, therefore, has a predesignated assignment for the required file response. Likewise, the captain will say "You're going to be the AB guy" to the first linesman, for example. Some people are really good at a task or perhaps they prefer to do airway management. So, they're the one on the team usually assigned to do that. Regardless, each day when they start,

everybody knows who's doing what at a cardiac arrest scene long before it happens. It's just as though you had a designated code team that day. Likewise, the respiratory therapist could be expected to do such a function, if up front everybody knows their job. By the way, I've applied this not only in the hospitals for cardiac care, but also for trauma care. When we have a bad trauma case come in, there's still an ABC kind of approach. It changes a little bit with a trauma patient, but it's the same general concept in our trauma resuscitations as well, even in the out-of-hospital setting.

Barnes: I think there needs to be a rotation on more than just mannequins. I think it would be completely reasonable for an anesthesiologist to say, "OK, I want you to intubate. I'm going to be here, but you put the tube in because you need the practice; if you're going to be good you need to get the experience—not just on mannequins, and not just in the operating room under controlled conditions, why don't you put the tube in this time?" or "Why don't you work with the nurse on the I.V. meds this time?" or "Why don't you take the defibrillator, even though someone else could do it. You need the experience on the defibrillator, so, on this particular day you're going to defibrillate."

Kaye: I agree, and we do that, too. Also, in the new program, there are 4 case-based scenarios stations. One station is respiratory arrest, with the universal algorithm and pulseless electrical activity. The second is V-fib—easy and difficult. That is, V-fib that responds to shock immediately and V-fib that doesn't. The third is asystole and bradyarrhythmias. The fourth station is tachycardias—stable and unstable. In the tachycardia station students learn cardioversion, but in each of the other 3 stations, there's a chance for each student to play all the roles, in-

cluding airway management, intubation, I.V. drugs, defibrillation, and monitoring. In other words, the new course is really a series of Megacodes. In the classroom, we need to stress the minimal roles needed and have each person play them. In the real situation, we should rotate people and assign them different roles, so they learn all of the specific skills.

Pepe: What Tom's (Barnes) saying is that you don't just want to be a teaching hospital, but also a learning hospital. Right?

Barnes: What I'm saying is that—if, for example, you have one respiratory therapist at the code and he usually manages the airway problems but needs experience with the defibrillator, there would be nothing wrong with a nurse doing the bag-valve ventilation, so the therapist has the opportunity to gain experience with defibrillation.

Kaye: Well, actually the nurse should do the bag-valve ventilation because the nurse should know how to do that too. The same is true for the residents because 2 codes may occur simultaneously.

Halperin: Very intriguing talk. I have a question about the studies you presented that showed no effect of ACLS training. There were 2 or 3 studies presented. Number 1, were the studies really big enough that they could show a difference?

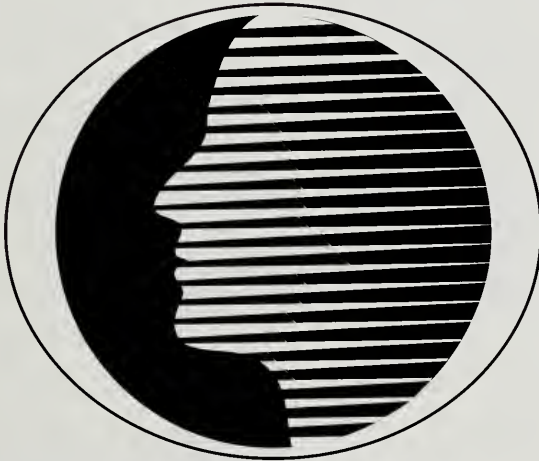
Kaye: No, it's really not good research, but there was no study that showed any benefit, which is very interesting.

Halperin: The other question is was there any control for any studying that the students might have done before the course? Because ACLS exists, some people may actually read the material prospectively. Was that taken into account?

Kaye: No, but then retention should be better, but it wasn't. Or maybe those who did well on their retention re-evaluation had reviewed the mate-

rials. Educational research, as you know, is very difficult. As they say in the literature, more work is needed. Now that we have a new ACLS pro-

gram, we really need to evaluate its effect on both learning as well as patient outcome.



FACE TO FACE WITH CHANGE

AARC 41st Annual Convention and Exhibition
December 2-5 • Orlando, Florida

Strategies for Maintaining ACLS Skills in Hospitals

Karen M Boudin MA RRT

- I. Introduction
- II. Training Issues
 - A. What Should an ACLS Provider Know?
 - B. What Skills Should the RCP Assume?
 - C. What Frequency of Retraining Is Necessary?
 - D. What Are the Methods for Maintaining ACLS Skills?
 - 1. Interactive Videodisc Technology
 - 2. Megacode Evaluation
 - 3. Simulated Cardiac Arrests
 - 4. Other Models
 - E. Should ACLS Training for RCPs Be Voluntary or Mandatory?
- III. Clinical Issues
 - A. Can RCPs Improve Patient Outcome?
 - B. Is the Outcome of Nonphysician-Managed Resuscitations Different?
 - C. What Level of Training Is Necessary?
- IV. Financial Issues
 - A. Should Resuscitation-Team Members Be Paid More?
 - B. What Costs Are Associated with Maintaining ACLS Skills?
- V. Psychological Issues
 - A. Enthusiasm—How Can It Be Maintained?
 - B. Status—How Do We Improve It for RCPs?
 - C. Emotional Support—What Is Necessary?
- VI. In Summary: Current Experience with ACLS Training

Introduction

Respiratory care practitioners (RCPs) have been valuable and useful members of the emergency resuscitation team since the emergence of inhalation therapists in the

Ms Boudin is Education Coordinator, Department of Respiratory Therapy, Stanford Health Services, Stanford, California.

A version of this paper was presented by Ms Boudin during the Journal Conference, "Resuscitation in Acute Care Hospitals" held in Cancun, Quintana Roo, Mexico in October 1994.

The author has no financial interest in any of the products mentioned in the text.

Reprints: Karen M Boudin MA RRT, Department of Respiratory Therapy, HCO23, Stanford Health Services, 300 Pasteur Drive, Stanford CA 94305.

early 1950s, and important medical advances in the field of cardiopulmonary resuscitation (CPR) and emergency cardiac care (ECC) have occurred since then. Traditional basic life support (BLS) skills are no longer adequate resuscitation tools for first-responding hospital personnel; the principle of early defibrillation by BLS personnel is fast becoming the standard of care for patients in cardiac arrest.¹ Advanced cardiac life support (ACLS) training for respiratory care practitioners (RCPs) is necessary to maintain our value as primary members of the resuscitation (or code) team and a crucial step in our goal to solidify and expand our role in today's evolving health-care-delivery system.²

Despite the active participation of RCPs as primary responders to in-hospital resuscitations, I found few articles documenting the capabilities of the RCP in resuscitation or emergency care. Much of the information I present is, therefore, based on studies of emergency-department personnel (ie, resident physicians, interns, medical students,

and nurses). I hope that this paper stimulates more research by RCPs on an important topic in which a great deal of research is needed! Unless we document and market our ability to function at an advanced clinical level, our role will fail to develop properly.²

In this paper, I present issues concerning ACLS skills with particular attention to the hospital-based RCP's ability to maintain these skills. Several key questions relating to training, clinical, financial, and psychological issues are addressed. Although no data are available on the number of RCPs who provide ACLS, my impression is that the number is small compared to the total number of RCPs practicing in hospitals. Based on the literature, this paper might have been more appropriately titled "Strategies for Obtaining ACLS skills." In an attempt to publish a more balanced account of the RCP's involvement in ACLS, I contacted several respiratory care departments whose administrators appeared to have a progressive attitude towards ACLS training. Several of these respondents³⁻⁵ expressed support of the recommendation by Barnes and Durbin⁶ for mandatory ACLS training in letters published in this journal. Thus, the last section of this paper focuses on the current ACLS experience of 8 respiratory therapy departments. ACLS training can lead to skill acquisition and the potential for role expansion as evidenced by several of these RCP managers who have shared their experiences.

ACLS training is the single most critical factor in properly establishing the RCP's role in emergency care,² and, as this role evolves, we must continually look for new ways to contribute to patient care in a positive and cost-effective manner.

Training Issues

What Should an ACLS Provider Know?

The American Heart Association (AHA) ACLS Instructor's Manual⁷ clearly states the two learning objectives of an ACLS course: first, the evaluation and management of the first 10 minutes of a witnessed cardiac arrest in an adult in ventricular-fibrillation, and second, the critical actions contained in the "core" ACLS cases (Table 1).

Table 1. The Nine "Core" ACLS Cases

- | |
|---|
| • Respiratory arrest |
| • Witnessed ventricular fibrillation |
| • Refractory ventricular fibrillation/pulseless ventricular tachycardia |
| • Pulseless electrical activity |
| • Asystole |
| • Acute myocardial infarction |
| • Bradycardia |
| • Unstable tachycardia |
| • Stable tachycardia |

What Skills Should the RCP Assume?

The actual performance of the critical actions described in the ACLS cases collectively rests on the shoulders of various resuscitation-team members. Barnes and Durbin⁸ suggest that RCPs should assume several critical tasks when they are the best trained responders at the scene. The AARC Clinical Practice Guideline for resuscitation in acute-care hospitals⁹ includes the RCP as one of the members responsible for assuming a number of ACLS tasks (Table 2).

Table 2. Resuscitation Responsibilities for RCPs

- | |
|---|
| • Assessment of patients for respiratory and/or cardiac arrest |
| • Activation of the resuscitation team |
| • Administration of BLS techniques |
| • Provision of mouth-to-mask ventilation |
| • Attachment of ECG and automatic defibrillator electrodes |
| • Attachment of pulse oximeter and capnograph |
| • Provision of a written report of the resuscitation effort |
| • Moving resuscitation equipment to the scene |
| • Collection of arterial blood for analysis |
| • Advanced ECG monitoring and dysrhythmia recognition |
| • Tracheal intubation |
| • Defibrillation and cardioversion with automatic and manual electronic defibrillators |
| • Use of continuous and transport mechanical ventilators |
| • Use of manual or automatic chest compressors |
| • Preparation and administration of cardiac drugs |
| • Stabilization of patients in the post-arrest period |
| • Provision of access for rapid administration of intravenous fluids |
| • Managing ventilation via transtracheal catheter and cricothyrotomy |
| • Emergency treatment of tension pneumo- or hemothorax, with large-bore needle |
| • Interpretation of hemodynamic data |
| • Preparation of patients for emergency transport |
| • Evaluation of oxygenation, ventilation, and acid-base balance from blood gas reports. |

What Frequency of Retraining Is Necessary?

That resuscitation skills retention declines rapidly over time is well-documented.^{10,11} Skills that are not used regularly soon after they have been learned do not become part of an individual's repertoire.¹² The performance of safe defibrillation deteriorated rapidly for nurses within 6 months of successful completion of the course.¹³ Frequent intubation errors were identified in medical students 2-3 months after initial training.¹⁴ Walters¹⁵ reported that ambulance staff showed a good knowledge of the ACLS treatment protocols, but errors in applying skills were common. The authors believed that improvement in these skills would have optimized the lifesaving capabilities of the ambulance staff, but because the staff received no feedback, they were probably unaware of how they might improve their performances.

The assumptions are that competency is maintained by performing a large number of procedures⁵ or by performing the skill a minimum number of times within a specific time frame.¹⁶ Evidence suggests that staff who participate in resuscitation events on a regular basis do not retain their knowledge or skills to any greater degree than those who participate less frequently or never.¹⁷⁻¹⁹ This may be due in part to the effectiveness of instruction. Poor retention may be explained by the student's inability to acquire or master the skills in the first place for a variety of reasons.^{17,20} Often, instructors have not taught in a standardized way, practice time has been limited, and errors in performance have not been corrected.²¹ One author recommends an initial review as early as 2-4 weeks after training, with short reviews every 3-6 months.²² The AHA recommends frequent practice sessions to enhance skill retention, with full ACLS retraining every 2 years.²³

The question, What frequency is best? cannot be answered simply; there is no magic period of time that elapses between competency and incompetency. However, some guidelines can be used to help hospital respiratory care departments establish a retraining program. (1) ACLS providers should retrain as often as needed, based upon their role in resuscitation;²⁴ therefore, identification of the skills that require reinforcing is first and foremost. (2) The current ability and/or performance level of the staff must be determined. Monitoring of staff is, therefore, necessary to assess the adequacy of the initial training program, to evaluate how training translates into actual practice, and to identify aspects in which retraining is required.¹⁵ All clinical staff should be monitored for skills and knowledge deficits, even those who participate regularly in resuscitations. (3) The optimal method for retraining must be determined. Mannequins or plastic models may be all that is needed for maintaining BLS skills (ie, rescue breathing and chest compressions). Access to supervised intubations on animal models or live patients is recommended for intubation retraining. (4) Respiratory care department educators and managers must consider the costs involved with the method of retraining for the number of those requiring retraining. The cost is likely to be a key factor in determining the frequency with which retraining is conducted.

What Are the Methods for Maintaining ACLS Skills?

To retain proficiency in ACLS psychomotor skills (and decision making), rescuers need regular retraining or refresher sessions.²⁵ Individualizing these sessions to meet the specific needs of a group is recommended.¹² Pretesting can be used to identify individual practitioner problems as well as group deficiencies. Improving the performance of the individuals should be the goal of any method employed

to maintain ACLS skills. A variety of methods are described in the literature including interactive videodisc technology (IVD), Megacodes, simulated cardiac arrests (SCAs), and other living and nonliving models.

Interactive Videodisc Technology. Educators in medical education must continually search for novel and effective ways to teach and assess learning. The application of computer technology as an innovative educational tool is quickly becoming commonplace. An interactive videodisc system can be defined as a computer-based system that employs a laser videodisc player to enhance the presentation by providing realistic audiovisual effects (Fig. 1²⁶). IVD is interactive because it adjusts instructional sequences in response to learner input.²⁷ In 1980, The AHA National Center and the Committee on Emergency Cardiac Care began the design of an IVD system for teaching BLS; by 1983 validation studies confirmed its ability to train and test people in CPR competence.²⁸ In January 1989, the AHA accepted the Actronics course (Interactive Multimedia Courseware, American Heart Association, Dallas TX and Actronics Inc, Pittsburgh PA) as a valid alternative



Fig. 1. Photograph depicting a clinician performing chest compression on a resuscitation mannequin, that is connected to an interactive videodisc system. Note the pulse waveforms on the screen. (Courtesy of the American Heart Association & Actronics Inc.²⁶)

for ACLS course completion.²⁹ The system was originally developed to increase the number of persons trained in BLS and ACLS while providing a standardized course of high quality that did not require an increased number of instructors.²⁸ Numerous benefits have been reported from this type of system including ease of operation, 24-hour availability, objective and consistent instruction, fair and impartial scoring, and employee enthusiasm.³⁰

The ACLS courseware series presents 14 hours of instruction in 4 modules, each with a test station. Because of its interactive nature, the system actually tailors the content of the course to respond to each student's individual needs. For example, if the student's actions are correct, the system commends the student. If not, it corrects the student, indicating the error and showing and explaining the right way to perform the procedure.²⁶

The 4 modules, Arrhythmia Recognition, Airway Management, Megacode, and Resuscitation Adjuncts and Automated External Defibrillation, are available individually, allowing implementation in stages. Together, the modules satisfy the AHA's requirement for BLS and ACLS training and formal validation.^{26,27} Because the AHA's CPR/ACLS Learning System is considered an extension of the training network, a national AHA course completion card is issued to acknowledge that the student has satisfied the guidelines for successful course completion as determined by the national AHA Committee on ECC.²⁸

A study comparing IVD instruction with an ACLS instructor in the testing of airway management skills suggested that the IVD system is an acceptable adjunct method for instruction and testing; however, it cannot be considered suitable for stand-alone instruction.²⁹ Disadvantages specific to IVD are machine malfunctions and the inability of the sensor to detect poor performance. In subsequent research, Rubens³¹ found that students prefer the presence of an instructor or facilitator during practice and instruction sessions. Other computer-aided instructional products designed to teach ACLS skills (ie, Cosmic Osmo, Code Red) have been described in the literature.^{32,33}

Megacode Evaluation. The Megacode teaching and evaluation station is well-known to past and current ACLS providers. Participants practice as members of a resuscitation team and learn to integrate the knowledge and skills of ACLS.^{10,34} Team roles typically include team leadership, airway management, chest compression and intravenous-catheter placement, drug administration, and record keeping; these are summarized in Table 3.³⁵ When Megacode is used as a training tool, participants are evaluated in each role during a simulated arrest using case scenarios. When it is designed as an evaluation tool, students are individually evaluated while playing the role of the team leader.³⁶

Table 3. Roles and their Functions during Megacodes³⁶

	Role	Function
Student		
I	Code Leader	Supervises and directs the resuscitation Diagnoses the cardiac rhythm
II	Airway Management	Establishes a patent airway and initiates ventilation
III	Chest Compression	Maintains blood flow by simulating heart-muscle contraction Monitors pulse as warranted
IV	Intravenous lifeline	Establishes intravenous access Administers drugs Keeps resuscitation record
Faculty		
A	Instructor	Operates dysrhythmia generator Summarizes clinical scenario for students Directly evaluates code leader's performance
B	Instructor	Supervises and evaluates airway management, ventilation, and assessment Safety director
C	Instructor (optional)	If available, supervises and evaluates Students III and IV

The Megacode method can be used for the purpose of refresher training to ensure that competency in ACLS knowledge and skills is maintained.³⁴ Retraining is based on the assumption that either the information was never learned in the first place or has been totally forgotten.¹² Regardless of the reason for the lack of skills, it is best to identify deficient areas that can be specifically addressed during retraining, so that retraining can be tailored to each individual's or group's specific needs.

Simulated Cardiac Arrests. The terms simulated cardiac arrest and Megacode have been used interchangeably in the past by many authors. However, a recent study³⁷ described SCAs as a method to determine the ability of the resuscitation team to respond to an emergency call in a timely manner. Other studies³⁸⁻⁴⁰ describe SCAs as a method of monitoring resuscitation performance on a multidisciplinary level. I believe this strategy is distinctly different from the previously described Megacode and merits consideration as a separate method for ACLS evaluation and retraining.

SCAs employ the element of surprise. They have all the components of a Megacode but are unscheduled events and the participants vary depending upon the assignment of code beepers at the time the SCA is announced.⁴¹ Therefore, individuals can only be monitored randomly,

eliminating SCA as the preferred method of evaluation for a specific individual or group. The main objectives of SCAs are to introduce hospital personnel to a cardiorespiratory arrest situation, identify deficiencies in the current system, reinforce ACLS education, and exchange ideas.^{37,39} Typically the exercise consists of placing a mannequin on a bed or in the corridor and asking the nearest bystander to react exactly as he or she would if the mannequin had been a patient or visitor who had collapsed.³⁸ This type of retraining strategy can be extremely valuable in identifying systems and performance deficiencies (ie, equipment, communications, supplies, and team response). SCA videotapes allow the participants to observe their own mistakes, an experience that creates a good setting for constructive criticism from peers and instructors.⁴¹

Other Models. Models for teaching lifesaving skills and technical procedures have been used for decades. A variety of them have been described in the literature, each with inherent advantages and disadvantages in terms of availability, cost, and efficacy. Often, legal and ethical issues must also be considered.

Living Patients. The use of live patients is common for teaching intubation skills. Many respiratory therapy departments with intubation responsibility have described clinical rotations through the operating room that are arranged as one component of the intubation training program.^{5, 16,42-44} Supervised by an anesthesiologist or his designee, each trainee is required to perform a specific number of successful endotracheal intubations on anesthetized patients. Whereas didactic training and practice on mannequins is relatively easy to coordinate, some believe that these methods alone are inadequate to ensure competence without additional training and practice on patients who are anesthetized.⁴⁵ When access to this type of clinical training is limited, a method for identifying only those particular RCPs with substandard performance should be adopted.

When practicing skills involving assessment, learners are encouraged to use themselves and each other as live models.⁴⁶ An unexpected benefit—increased empathy—was reported by Nelson⁴⁷ when students practiced the procedure for obtaining an arterial blood-gas sample on each other. Barrows⁴⁸ describes the explosion of activity that has occurred in the use of standardized patients (SPs) as an assessment tool for clinical competence. The term, standardized patient, is used to define both a simulated patient (a well person trained to simulate a patient's illness in a standardized way) and an actual patient (trained to present his or her own illness in a standardized way.) Readers with further interest in the value of SPs, should review the *Proceedings of the Association of American Medical College's (AAMC) Consensus Conference on the Use of Standardized Patients in the Teaching and Evaluation of*

*Clinical Skills.*⁴⁹ Also, an article by Vu⁵⁰ describes 6 years of experience using SPs.

Deceased Patients. Although few reports are available regarding the practice of using recently deceased patients for training ACLS skills, it is probably a common teaching method. In a survey sent to 5,397 emergency departments in the country, 537 of 992 respondents confirmed the use of recently deceased patients for intubation training.⁵¹ This practice has been recently criticized on religious, legal, and ethical grounds. Iserson⁵² raises 4 questions that should be resolved regarding this method of instruction: (1) Is it ethically and legally permissible to practice and teach noninvasive and minimally invasive procedures on newly deceased emergency-department patients? (2) What are the alternatives or possible consequences of not practicing noninvasive and minimally invasive procedures on newly deceased patients? (3) Is consent from relatives required? (4) Should doctors in emergency departments allow or even encourage this use of newly deceased patients?

Providing health-care professionals with a closely supervised intubation practice using newly deceased infants has been described by Benfield et al.⁵³ The AHA supports this practice, stating that it is ethically justifiable in that it is nonmutilating, brief, beneficial to others, and an effective teaching technique.⁵⁴

Cadavers. The use of cadavers for teaching anatomy is well established in medical schools. Their use for teaching ACLS skills has been described by Nelson.⁵⁵ Because cadavers are the bodies of people who have given specific consent for their bodies to be used for teaching or research, the ethical issues are minimal. Almost every technique used for airway management can be well-taught with the cadaver model; the exception, of course, is mouth-to-mouth rescue breathing.

Animals. Animal models have been used to teach intubation skills.^{14,51} Although anatomic differences often make them a poor choice, they provide a degree of realism during performance of certain skills. Training programs for intubation of neonates have been described that use kittens for intubation because the kitten's larynx is very similar in size and anatomy to that of the human infant.⁵⁶ The use of animals is a controversial topic in today's society with animal support groups becoming increasingly vocal and influential in their demands to reduce the number of animals used in research and education.

Artificial Models. Plastic models such as CPR mannequins, intubation heads, and arms for venous and arterial punctures are popular training tools. The most recent models are often well-designed and accurately reflect human anatomy. Accessories such as artificial blood are available to add realism to the use of these devices for training. Despite their initial expense, artificial models are durable and can be used to train a large number of students for many years.

Should ACLS Training for RCPs Be Voluntary or Mandatory?

In June 1992, *RESPIRATORY CARE* published an editorial by Barnes and Durbin⁶ who advocated ACLS training for those RCPs who respond to resuscitations, cover the emergency room, work in critical care units, and serve on transport teams. Strong support of their ACLS-mandate editorial was apparent by the number and content of letters responding to the editorial;^{3,4,57-60} with several respiratory care department managers describing their accomplishments in providing ACLS training.

In December 1992, the president of the AARC made a recommendation to the AARC Board of Directors to compose a position statement advocating voluntary, not mandatory, ACLS training.⁶¹ This recommendation was based on opinions solicited from the AARC membership and the majority of respondents were not in favor of mandatory ACLS training. Acceding to the majority, President Demers suggested that a heightened awareness of the need for ACLS training for RCPs might result from the statement so that mandatory ACLS training might become a reality in the future.⁶¹

I believe the future is now. As primary members of the resuscitation team, our daily activities demand proficiency in the essentials of ACLS, not only for our profession's benefit, but for the patient's benefit! Resuscitation is a team effort. No one head is as great as all heads together. All members must understand the role of each team member and all should have a common understanding of the algorithms used to manage patients found in cardiac arrest or in impending arrest. ACLS training may be the single most critical factor in properly establishing the RCP's role in emergency care² and is the pathway for assuming greater responsibilities during resuscitation.

I believe that clinically active respiratory care managers should complete ACLS training to serve as role models and advocate for their staff's participation in ACLS training programs. I further believe that hospital-based RCPs should become ACLS Providers to improve patient care and to participate on an equal basis with nurses in resuscitations. And last but not least, I believe that educators should become ACLS Instructors to increase the instructor pool and to provide the training and preparation necessary for students and practitioners to succeed in ACLS training programs.

Clinical Issues

Can RCPs Improve Patient Outcome?

In performing ACLS, it is the response time and the quality of the intervention that ultimately affect long-term survival from cardiac arrest.⁶² There are data to suggest

that RCPs as primary responders to in-hospital resuscitations have an opportunity to improve patient outcome. A recent study³⁷ published in *RESPIRATORY CARE* reported response times of a hospital arrest team. In order of appearance, the first 3 members of the cardiac arrest team to arrive were the physician, RCP, and ICU nurse (Fig. 2). The difference in median response time (MET) of the physician (3.17 min) and respiratory therapist (3.20 min) was 0.03 minutes; the MET of an ICU nurse (3.58 minutes) was 0.41 minutes after the physician.

Outcome is also dependent on the quality of intervention. Little is known about the effects of the quality of ACLS on patient outcome; however, a recent study by Birnbaum et al¹³ suggests that training directed to the entire team likely to participate in the provision of ACLS favorably affects the overall practice of ACLS and the survival rate of patients with ischemic heart disease. Respiratory therapists improved their overall performance after an ACLS training program as documented by an increase in adjusted mean test scores obtained before initiation and on conclusion of the training period. The course included 39 hours of instruction time; each participant was required to successfully participate in complex simulations that required each to integrate the materials and skills acquired in the course, including performance in the team leader role.

Is the Outcome of Nonphysician-Managed Resuscitations Different?

"Is there a doctor in the house?" This classic phrase implies a belief that with the arrival of a physician comes the medical knowledge needed to handle any emergency.⁶³ Can we assume that all physicians are knowledgeable and competent in managing cardiopulmonary emergencies? Kaye and colleagues⁶² described 3 studies that documented poor performance of ACLS by physicians. ACLS training programs in rural community hospitals are often minimal.⁶⁴ Sanders et al⁶⁵ reported the efficacy of an ACLS training program in which none of the physicians in the community were certified in ACLS prior to the study. It is conceivable that little advantage may be gained from a physician-managed resuscitation when compared to one that is not, if the physicians have less formal training in resuscitation than other health-care providers on the team. Regardless of the team leader's credential, ACLS training can have a positive effect on performance and may improve patient care and survival.¹³

No studies are available on patient outcome from in-hospital resuscitations that specifically utilize nonphysicians as ACLS team leaders. However, nonphysician resuscitation-team models are widespread in prehospital and inter-hospital transport care. Emergency medical technicians (EMTs) have been trained to intubate and defibrillate patients, and

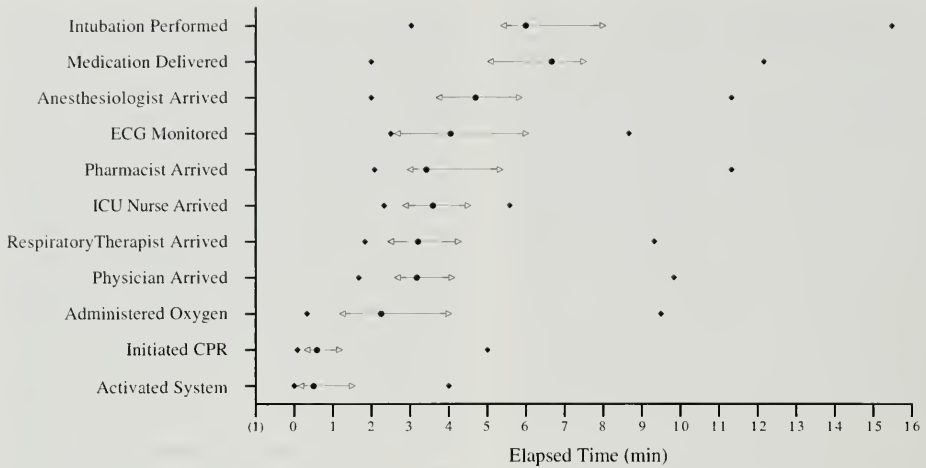


Fig. 2. The time in minutes for the measured events are shown as median •, interquartile range—the lower and upper 25th percentiles (<—), and the full range—the lowest and highest value (•—).

their effectiveness has been confirmed in several controlled evaluations.⁶⁵ Critical care transport flight teams that utilize nurse-nurse or nurse-RCP staff combinations are highly trained (ie, as providers or instructors in ACLS, advanced trauma life support [ATLS], pediatric advanced life support [PALS], and neonatal resuscitation program [NRP]) and authorized to manage resuscitations for all patient populations under institutional protocols. In these cases, qualified resuscitation-team members may achieve the same level of success in the absence of physicians.

What Level of Training Is Necessary?

There is no such thing as ACLS certification. True or False? ACLS courses have dropped the pass/fail policy. True or False? Both statements are true. Dramatic changes in the ACLS training program were published in May 1994 by the ACLS Education task force.⁶⁶ Most obvious was the change from a subject-based approach to a problem-oriented, scenario-based educational experience. Less obvious but more significant was the refocus of the purpose of the ACLS course: to help all course participants succeed in acquiring the skills and knowledge they need for their professional role in resuscitation attempts. The term, refocus, is important. From the perspective of the AHA, the emphasis on certification, pressure to obtain a "card", and focus on passing or failing was a perversion of the original goals.

The renewed educational philosophy discourages the use of the term, certification, and recommends "course

completion" as the preferred terminology. As early as 1979, the ECC Committee of the AHA took steps to clarify the intent of successful course completion.²⁴ This intent was published in the 1980 JAMA supplement⁶⁷ stating that the term, certified, is equivalent to successful completion. Beginning in 1989, the AHA removed "certification" from all the cards and materials used in the BLS, ACLS, and pediatric resuscitation programs.⁶⁸ Despite these efforts, widespread reference to ACLS "certification" continues among health-care professionals. The goal of the AHA is not the certification of clinical competency²⁴ nor is the ACLS Provider's Course meant to license, certify, credential, or warranty providers.⁶⁹

The question of competency remains. The fact that competency should be measured is indisputable. By whom and how are more difficult questions. Clinical competence should be tested by the entity held accountable for the quality of the care delivered by its providers.⁶⁸ The burden of proof essentially is on the employer, certainly not the AHA. The 1994 standards of the Joint Commission on accreditation of Health Care Organizations have placed a significant emphasis on clinical competencies for respiratory care practitioners.⁷⁰ Competency evaluation of resuscitation skills and knowledge has now become the responsibility of individual departments. Competency standards may vary according to job descriptions and roles and must be clearly stated in the department's policy and procedure manual. Ideally, competency assessment should be incorporated into the department's quality assurance, continu-

ous quality improvement, or performance improvement program. With the introduction of objective measurements and regular evaluation or monitoring of ACLS skills, a program can be designed to ensure competency and provide retraining as necessary.

Financial Issues

Should Resuscitation Team Members Be Paid More?

Institutional differences pose a great number of variables when determining whether monetary compensation can or should be provided for staff with ACLS skills. Staff who voluntarily complete ACLS training or become ACLS Instructors should be recognized for their effort and professional attitude. Various methods that have been used include: (1) increased performance appraisal scores that could potentially result in a higher raise, (2) a salary increase, and (3) increased wages by advancement or promotion to a job with more responsibility.

What Costs Are Associated with Maintaining ACLS Skills?

In these times of cost constraints, educators who are charged to ensure competency are faced with a real dilemma. Emergency resuscitation equipment is expensive! Costs vary depending upon the methods used and the number of professionals whose competency must be maintained. Costs that should be considered include (1) time away from direct patient care, (2) instructor preparation and training, (3) resuscitation supplies and equipment, (4) model access and/or maintenance, and (5) ACLS course costs for regular renewal. Actronics Inc offers ACLS and BLS justification worksheets in their information package. By simply inserting your institution's facts and figures, you can (1) determine your present costs for an internal or external ACLS course, and (2) project AHA-system costs. The worksheet is intended to demonstrate net savings over a 4-year period.

Psychological Issues

Enthusiasm: How Can It Be Maintained?

RCPs must be able to use their acquired knowledge, skills, and talents in order to maintain enthusiasm as a valuable member of the resuscitation team. Frustration is a natural reaction in situations in which health-care providers are not allowed to perform in roles for which they are most qualified. Physician advocacy and medical director support may be crucial to role expansion, especially when data to show that RCPs can participate in a timely,

safe, and cost-effective way are lacking. If RCPs are ready to accept the challenge, performing intubations, defibrillation, electrocardiographic monitoring and insertion of arterial lines and intravenous catheters are all within our scope of practice and depend only on the willingness of the medical staff at each facility to approve.

How Do We Improve the RCP's Status on the Team?

The first step begins with ACLS Provider training, ideally in a multidisciplinary format. Not until we can be recognized as knowledgeable in all aspects of cardiopulmonary resuscitation can we achieve parity with other members of the team. We must demonstrate through our knowledge, skills, attitudes, and behavior, that we are professionals who deserve recognition.

What Emotional Support Is Necessary To Deal with Reactions to Unsuccessful Resuscitations?

It is important to realize that caregivers as well as families experience grief reactions. Health-care providers may experience additional emotions (ie, anger, confusion, or guilt) after unsuccessful resuscitations. The new ACLS training program includes a case entitled "Psychological Effects of CPR on the Rescuer," which includes recommendations for critical incident debriefing.⁷¹ The critical incident debriefing is a discussion, led by the resuscitation leader, that should follow any resuscitation attempt in which there appeared to be significant anxiety, confusion or other emotional overtones, as well as after unsuccessful resuscitations in which the death was unexpected.

Hospitals are now required to have advisers such as ethics committees and bioethicists who are available for clinical ethical consultations.⁷² These committees or consultant members are excellent resources for situations in which health-care providers need further follow-up after the critical incident debriefing.

Current Experience with ACLS Training

The scope of the issues discussed in this paper vary among hospitals according to bed size, operating budget, commitment to medical education, illness severity of inpatients, and other factors. The following summaries have been written from the reporting practitioner's perspective, so that you may realize more fully the impact of their experience and be helped by it. I began my solicitation of testimonials from the authors of the letters responding to the ACLS-mandate editorial by Drs Barnes and Durbin.⁶ They, in turn, referred me to other hospitals with respiratory therapy departments that were actively involved in

ACLS training. Therefore, these 8 centers represent a convenience sample. Each was asked to respond to a discussion-type standard questionnaire. Subsequent follow-up was necessary to ensure accuracy.

Duke Medical Center—

Durham, North Carolina

Janice Thalman BS RRT

Our pediatric-, neonatal-, and adult-care medical center is licensed for approximately 1,000 beds. The Respiratory Care Department was established in 1965, originally under the Department of Anesthesiology. At that time, we offered to carry the intubation equipment to all resuscitations and thus began our intubation service. Over the years, our track record has been good. Training is now formalized; currently our airway-team members are registered respiratory therapists, have passed intubation certification, and have completed the ACLS and PALS courses.

Our department is composed of 109 clinical full-time-equivalent employees (FTEs)—Level I (intermediate care only) and Level II—10 supervisors, and 20 ECLS (extra-corporeal life support) specialists. The RRT credential must be achieved to advance to Level II; the practitioner selected for intubation training must be a supervisor or Level-II therapist and must complete instruction in ACLS. Pediatric practitioners are required to have satisfactorily completed PALS. Supervisors maintain ACLS-Instructor status and respond to all resuscitations. ECLS specialists are trained in ACLS and PALS. The Emergency Department is staffed 24 hours/day with an RCP who has completed ACLS and PALS. Six RCPs/shift are available to respond to resuscitation calls. Although staff are not paid more for ACLS or PALS course completion, the Level-II RCPs are at a higher salary scale, than the Level I staff.

The RCP's role in emergency care includes intubation, basic life support (traditional airway care and chest compression), nasogastric-tube insertion, endotracheal drug administration, arterial punctures, and physician instruction in these procedures. Our emergency response is unique in that emergency airway equipment is handled only by the respiratory care airway team. We stock 8 portable emergency bags instead of providing airway equipment for all emergency carts throughout the medical center. Respiratory Care Services performed 791 intubations over a 1-year period, and RCP staff members maintain records for all intubations performed. Additionally, the respiratory therapy supervisors are the primary data collectors during resuscitations, formerly a role of the Medicine fellow. This information is provided to the hospital's Code 5 Committee for quality assurance purposes.

RCPs participate in ACLS and PALS courses conducted internally by the Respiratory Care Department. Classes are held twice a year, and instruction is provided by the supervisors and by our medical director who also supervises the Megacode. Intubation certification includes 4 hours of classroom training, 2 hours of clinical simulation, a written examination, and 10 successful intubations (under supervision). Successful intubations are those in which the trachea is intubated with fewer than 3 attempts and without complications. Maintenance of skills is documented; each intubator must perform a minimum of 6 intubations/year within standards or they must repeat the intubation course. As observers at each intubation, the area supervisor provides a critique of the intubation following each procedure.

Intubation training costs are based on 8 hours of average pay per clinical FTE annually for approximately 60 FTEs. Additionally, 2-4 RCPs may attend a 2-day Instructor course each year. The respiratory care ACLS Instructors are paid for their hours of instruction at their standard rate of pay. Course participants must pay \$10.00 for training manuals. The department has purchased an electrocardiogram-rhythm simulator for ACLS training; all other expenses for ACLS and intubation training are taken from the payroll budget.

Mayo Medical Center—

Rochester, Minnesota

Curt Buck CRNA RRT

Our 1,000-bed medical center includes 2 hospitals with pediatric, neonatal, and adult care and a fixed-wing transport service covering a 5-state area. The Respiratory Care Department employs 126 (85 non-supervisory) personnel that are utilized in 9 ICUs and who participate on nurse-therapist interhospital transport teams. Established in 1968 by a CRNA/RRT, the Respiratory Therapy Department was instrumental in early CPR education at the Mayo Clinic. The lead-therapist position was created in the mid-1970s for a limited number of exceptional RRTs, considered to be the 'answer persons' for respiratory care. In addition to routine responsibilities, they perform intubations and arterial line insertions. During resuscitations, they provide basic life support and serve as secondary intubators when critical-care resident physicians are unsuccessful. The neonatal RRT-RN transport team was formalized in the early 1980s; now there is a RRT-RN pediatric team as well. The adult transport team utilizes RRTs when patients are intubated or when they are critically ill with the potential to require airway management.

The Respiratory Care Department is comprised of several clinical levels, with various credentials and AHA course completion requirements for each.

Clinical Level	Credential	AHA Training
Technicians (equipment delivery and maintenance)	non RCP	BLS
Staff therapists (ICU and limited floor care)	CRTTs & RRTs	BLS (ACLS encouraged)
Lead therapists	RRT	ACLS
Adult transport team therapists	RRT	ACLS
Pediatric and neonatal transport team therapists	RRT	ACLS & PALS & NRP

The RCPs respond to all resuscitations and emergency calls, which average 240/year. Our responsibilities include intubations, basic life support (traditional airway care and chest compression), and arterial-line insertion. We have developed various strategies for maintaining these skills: (1) Staff and lead therapists complete mannequin intubation competency checks annually and ACLS training every 2 years. (2) Transport team members must document airway management techniques used in each transport. They receive evaluation of the skills used during the previous quarter's transports. Operating room intubation experience is provided for those not meeting a minimum of 4 intubation/year and for retraining; an animal (pig) intubation practicum is available quarterly. (3) Pediatric and neonatal transport team members may attend a quarterly skills lab that includes animal intubation, central-line insertion, chest-tube insertion, and needle thoracotomy. Generally, the neonatal transport-team members are able to maintain these skills because of the high volume of procedures performed in the clinical setting. Quality assurance records of transport activity are collectively maintained by RCPs and the transport service. The respiratory care department takes part in the resuscitation quality assurance program through representation on the hospital's Resuscitation Committee.

The expense for ACLS training (\$80/person) is paid from the department budget, and training time is scheduled in the same manner as routine work assignments.

St Luke's Episcopal Hospital—
Houston, Texas
Elizabeth Bearden RRT

St Luke's Respiratory Care Department is responsible for coordinating ACLS classes within our 939-bed institution. It all began in January 1990 when two ACLS instructors discussed the need for a formal ACLS training program. This team of 2 persons consisted of a respiratory

care educator and a cardiology fellow. With support from the American Heart Association and our medical director, I became the course coordinator, a job which had been assigned to various people over the previous 10 years. Currently (1995), we have 35 instructors represented by respiratory care clinicians and supervisors, nursing clinical specialists, nursing educators, cardiology fellows, emergency-medicine physicians, staff physicians, pharmacists, and paramedics. In 1994, we offered 4 Provider courses and 1 Instructor course and plan to offer 5 Provider courses in 1995.

Our department has a staff of 66 therapists, comprised of Level-I and Level-II practitioners, supervisors, and clinicians. CRTTs and RRTs are all trained and assigned the same work duties; however, only RRTs are eligible to advance to the positions of relief supervisor, supervisor, and clinician. RCPs attend all resuscitations (average 0-5/week); their role includes basic life support (traditional airway care and chest compression), and arterial puncture. One strategy employed for maintaining skills relies on peer feedback from either fellow RCPs or other health-care professionals. When an RCP has been reported to perform poorly during a resuscitation, he/she undergoes a review of airway-management, basic-CPR, and other resuscitation skills. Although ACLS training is not required, RCPs are encouraged to complete ACLS training, and, whereas the greatest number of ACLS course participants are physicians and nurses, we have seen an increase in the number of RCP registrants since our department became responsible for the courses.

RCPs pay their own ACLS course registration fee and are reimbursed upon successful completion. Although ACLS-trained RCPs are not paid more than those who are not ACLS-trained, obtaining ACLS-Provider status affects their yearly performance evaluations and may impact their wage increases. The Respiratory Care Department has purchased and maintains all the equipment for ACLS courses with the exception of defibrillators and AEDs, which are provided by Nursing Service.

Santa Barbara Cottage Hospital—
Santa Barbara, California
Paul Sherman MS RPFT RRT

Our center is a 450-bed pediatric-, neonatal-, and adult-care center. Five years ago, ACLS Instructors and training-center coordinators were all nurses. Since that time, the involvement of a respiratory therapy ACLS Instructor and inclusion of ACLS in the RCP job description has enhanced the status of the respiratory therapists at our hospital.

The Respiratory Care department is comprised of the following levels, credentials, and AHA-course completion requirements:

Level	Requirements
Adult Coordinator	RRT, ACLS
Senior RCP	CRTT*, ACLS
RCP II	CRTT, ACLS
RCP I	CRTT, BLS
Ped/Neo Coordinator	RRT, PALS, NRP
Senior RCP	RRT-eligible, PALS, NRP
RCP II	RRT-eligible, PALS, NRP

*CRTT, certified respiratory therapy technician.

RCPs respond to all resuscitations (approximately 1 every other day). Our responsibilities include intubations, basic life support (traditional airway care and chest compressions), and arterial punctures, blood drawing from arterial lines, and analyses of blood samples. Although RCPs do not have primary intubation responsibility, we can intubate if no one else is qualified or after the resident has failed. One to two respiratory care staff members participate on the hospital's CPR Committee at all times; all resuscitations are reviewed by this committee.

Within the neonatal-pediatric department, there are 3 PALS instructors and 3 NRP instructors that teach the course 3 to 4 times a year. These RCPs respond to all codes in pediatrics, emergency room, neonatal and pediatric intensive care units, and newborn nurseries. We are the primary people responsible for airway management (including intubation) within the hospital and on transports. We have a nurse/RCP resuscitation team for the high-risk deliveries. In addition, RCP/nurse team members participate in a comprehensive, collaborative teaching and competency program.

Our strategies for obtaining and maintaining these skills include a comprehensive educational program and frequent Megacode practice sessions conducted by our department. Staff preparing to take the ACLS course are provided with a copy of "Study Guidelines for Passing ACLS," created by John Wadley RRT, our ACLS Instructor. They are asked to pair up with a partner to help them learn from their own individually prepared flash cards and study posters. Structured ACLS preparatory sessions are conducted that include a dysrhythmia review, use of an ACLS Instructional computer disk, sample test questions, and practice Megacodes. The ACLS course is free to employees of Santa Barbara Cottage Hospital.

Kodiak Island Hospital—

Kodiak, Alaska

Wayne Wallace BA RRT

Our 1-floor hospital has 44 beds and serves patients of all ages. As manager for Respiratory Care, Physical Therapy, Radiology, Laboratories, and the Safety

Department, I consider ACLS a job requirement for the respiratory care staff, which includes myself and 2 CRTTs. We assist the physician during exercise testing and must be able to manage emergencies, often assuming traditional nursing responsibilities because nurses are not ordinarily in attendance. We respond to all resuscitations (average 2-3/month).

The RCP's role in emergency care varies at Kodiak Hospital, and RCPs can perform all ACLS skills (ie, intubation, defibrillation and cardioversion, administration of intravenous and endotracheal drugs, and chest compression). Delegation of responsibilities follows the principle of availability—if everyone else is occupied and an RCP is available and qualified to intubate, then that RCP has intubation responsibility.

Our hospital administrators support AHA training for BLS, ACLS, PALS, and NRP courses and believe creating experts within the hospital is more important than sending staff 252 air miles to Anchorage for training. It is a value statement—several in-house staff are trained as instructors who then train the providers locally. I have become a PALS Instructor and an NRP Instructor, and I volunteer to teach courses for the nursing staff. Likewise, nurses teach ACLS and BLS courses for the rest of the hospital staff.

Training costs are absorbed by the hospital; staff are paid regular hourly wages while participating in or instructing an AHA course. Tuition fees are provided when necessary. Additional services include pastoral or mental-health debriefing. Our strategy for maintaining ACLS skills is to provide periodic retraining and retraining in response to substandard performance. Every resuscitation is reviewed by the Emergency Room/Intensive Care Unit Committee, and a respiratory care staff member participates on this committee.

Sinai Hospital—

Detroit, Michigan

Linda Zimcosky RRT

Sinai Hospital's Respiratory Care Department has coordinated the ACLS program since its implementation in 1978, in collaboration with Emergency Medicine Services. This was accomplished by the director who worked with the cardiologists to gain support for the development of an ACLS program. The roles of course director and medical director are currently performed by the chairman of Emergency Medicine. As Respiratory Care Services Administrative Director, my role is ACLS course coordinator. The 15 ACLS faculty are a combination of medical staff, respiratory care staff, and nurses. We offer 3 ACLS-Provider courses yearly, each with an enrollment of 70.

Our 603-bed hospital has a staff of 70 RCPs—the levels are Technician, Senior Technician, Therapist, Senior Therapist, Clinical Coordinator, and Supervisor. All levels must complete BLS training, and ACLS is highly encouraged but not required. Neonatal therapists are required to complete NRP.

The RCP's role in emergency care includes basic life support, rhythm interpretation, intravenous and endotracheal drug administration, and intubation when an anesthesiologist is not available. RCPs attend all resuscitations, which numbered 1,427 during the 1994 fiscal year. Our strategy for maintaining ACLS skills is to provide an opportunity for regular retraining in our ACLS courses. Obtaining BLS Instructor and ACLS Provider and Instructor status are recognized in the staff development section of an RCP's performance plan. Although staff are not paid more for ACLS course completion, an increase in their overall score for job performance may result in increased wages.

The Sinai Hospital Education Corporation has provided financial support for this program since its beginning in 1978. The fee of \$225 per participant for the ACLS/BLS course is waived for all hospital employees. The Respiratory Care Department has purchased and maintains all equipment for the ACLS courses.

Memorial Medical Center of East Texas—
Lufkin, Texas
David Henson BS RPFT RRT

The Respiratory Care Department at Memorial Medical Center is responsible for organizing and teaching ACLS classes countywide. All RCPs are ACLS Providers, and many are BLS and ACLS Instructors. The department's Assistant Director and I, the Director, are ACLS Affiliate Faculty members. This undertaking began in 1982—at that time, no RCPs were BLS-trained. A plan was formalized so that some RCPs would become instructors and teach BLS to the other RCPs. We soon began opening our classes to other hospital staff, and the plan was extended so that all RCPs became BLS Instructors, with a few RCPs as BLS Instructor-Trainers. In 1984, the grand plan expanded again—all RCPs were required to take ACLS, and encouraged to become ACLS Instructors. Successful ACLS course completion for 100% of our full-time staff was achieved in 1991. Although many RCPs are ACLS Instructors, they are actually not the largest component of instructors in Lufkin; we have many excellent nurses, paramedics, and physicians who help teach ACLS for the entire county.

Memorial Medical Center has 300 licensed beds, and the Respiratory Therapy Department has 20 full-time and 30 per-diem practitioners. We offer a 6-tier clinical ladder.

Tier	Requirement
1	Perform electrocardiograms
2	Perform floor therapy
3	Draw arterial blood
4	Perform intensive-care skills
5	Perform neonatal-pediatric care
6	Perform cardiopulmonary diagnostics

All staff members must be CRTT-credentialed with ACLS-Provider and BLS-Instructor status. Many are RRTs with training in PALS and NRP, and some have achieved ACLS-Instructor status. Full-time employees are sent to the first ACLS class scheduled after their hiring, and all temporary employees are scheduled within 1 year. RCPs attend all resuscitations and enjoy equal status with other members of the team. They function in all roles—airway management, intubation (with physician request), electrocardiogram-lead attachment and defibrillation, chest compression, arterial puncture, record keeping, and team leader. RCPs have achieved a great deal of community recognition and collegial appreciation from this endeavor. During a recent resuscitation, a physician observed an RCP functioning as team leader during a resuscitation. The following day, that physician personally commended the RCP for his performance.

Our strategy for maintaining skills is accomplished, in part, by teaching ACLS frequently, and by coordinating monthly 'mock codes' in all patient care areas. These are conducted jointly by staff from the respiratory therapy and nursing departments. Each group involved is graded on its performance in different categories and arrival times are recorded for various team members.

ACLS course registration fees, books, and instructor salaries are paid from the Respiratory Therapy Department budget. Staff receive paid education days for attending courses and a small wage increase for ACLS course completion.

Queen's Medical Center—
Honolulu, Hawaii
Danny Langston RRT

Our center is a 500-bed trauma center serving all types of patients. At the present time, our respiratory care staff enjoys a great sense of accomplishment and recognition from participating in the ACLS courses offered at our hospital. However, getting from there to here was over a difficult road with many obstacles. When I became the Respiratory Care Education Coordinator in 1991, RCPs were not allowed to take the ACLS courses; enrollment was limited to nurses, physicians, and mobile intensive care technicians (MICTs). After some insistence and persistence, the ACLS course director decided to allow RCPs to participate but

would not issue RCPs the AHA course-completion card. Support from a key Hawaii Heart Association physician and a nurse advocate was obtained and, together, we presented a proposal at an ECC meeting that resulted in a majority vote for RCPs to obtain ACLS provider cards. Back at Queen's Medical Center, a meeting of the Respiratory Therapy Medical Director, Respiratory Therapy Department Director, and Director of Nursing Education was held to negotiate the number of RCPs who could enroll in the ACLS-Provider courses. In the summer of 1993, RCPs received permission to attend and the preparation began as described below. We have a 100% pass rate and often receive the highest scores in the classes. With the exception of the technicians, ACLS training is now required for all RCPs at Queen's Medical Center. We also have several BLS-, ACLS-, and NRP-Instructors in the department at this time. The RCPs respond to all resuscitations. Our responsibilities include basic life support (traditional airway care and chest compressions), endotracheal drug administration, and cardiac-rhythm interpretation. The Respiratory Care Department offers a 3-level clinical ladder.

Level	BLS	ACLS	NRP	PALS	ATLS
Charge & Supervisor	X	X	X	X	X
Therapist (RRT)	X	X	X		
Technician (CRTT)	X				

Our initial strategy for obtaining these skills included an elaborate inservice program conducted by our department. We proceeded on this venture with the following educational plan:

Rhythm Interpretation

- Electrocardiogram videotape
- Electrocardiogram self-learning module (included ACLS algorithms)
- Electrocardiogram flashcards
- Electrocardiogram lectures at rounds
- Electrocardiogram pre- and posttest

ACLS Algorithms

- Guest lecture reviewing ACLS pharmacology
- Pharmacology lectures during patient-care rounds
- PROACT Project (Professional Resources Organized To Advance Clinical Training)
- ACLS flashcards
- Algorithm discussions at rounds

Each charge therapist (PROACT leader) took responsibility for 5 RCPs' instruction and understanding of 5 key ACLS algorithms. This included documentation of successful completion.

The ACLS course fee is paid by the hospital education fund, the Respiratory Care Department pays the therapist regular wages for attending the course. The staff members are responsible for purchase of the textbook, which is about \$10.00.

In summary, whereas many nursing administrators did not want us to participate in ACLS, many did, and they were the keys to our success. A 'nursing-satisfaction-with-RCPs' quality-assurance survey, conducted after our program was well underway asked the respondents to evaluate RCPs performance pre- and post-ACLS training. Improvement in the RCPs' knowledge and assertiveness in all categories was perceived by the nurses.

REFERENCES

1. American Heart Association: Textbook for Advance Cardiac Life Support. Dallas: American Heart Association, 1994:4-3
2. Kacmarek RM. The role of the respiratory therapist in emergency care. *Respir Care* 1992;37(6):523-532.
3. Bearden EF. ACLS—One department's experience (letter). *Respir Care* 1992;37(8):945.
4. Henson D. Dinosaurs, RCPs, and ACLS (letter). *Respir Care* 1992;37(8):946-947.
5. Thalman JJ, Rinaldo-Gallo S, MacIntyre NR. Analysis of an Endotracheal Intubation Service Provided by Respiratory Care Practitioners. *Respir Care* 1993;38(5):469-473.
6. Barnes TA, Durbin CG Jr. ACLS skills for the respiratory therapist: time for a mandate (editorial). *Respir Care* 1992;37(6):516-519.
7. American Heart Association. Instructor's Manual for Advanced Cardiac Life Support. Dallas: American Heart Association, 1994:5-1.
8. Barnes TA, Durbin CG Jr. Research on resuscitation by RCP's is long overdue (editorial). *Respir Care* 1994;39(7):712-714.
9. American Association for Respiratory Care. Clinical practice guidelines: resuscitation in acute care hospitals. *Respir Care* 1993;38(11):1179-1188.
10. Cavanagh SJ. Educational aspects of cardiopulmonary resuscitation (CPR) training. *Intensive Care Nurs* 1990;6(1):38-44.
11. Coleman S; Dracup K; Moser DK. Comparing methods of cardiopulmonary resuscitation instruction on learning and retention. *J Nurs Staff Dev* 1991;7(2):82-7.
12. Kaye W, Mancini ME, Rallis SF. Advanced cardiac life support refresher course using standardized objective-mega code testing. *Crit Care Med* 1987;5:55-60.
13. Birnbaum ML, Robinson NE, Kuska BM, Stone HL, Fryback DG, Rose JH. Effect of advanced cardiac life-support training in rural, community hospitals. *Crit Care Med* 1994;22(5):741-749.
14. Nelson MS. Medical student retention of intubation skills. *Ann Emerg Med* 1989;18:1059-1061.
15. Walters G, Glucksman E. Retention of skills by advanced trained ambulance staff: implications for monitoring and retraining. *BMJ* 1989;298(6674):649-50.
16. Zyla EL, Carlson J. Respiratory care practitioners as secondary providers of endotracheal intubation; One hospital's experience. *Respir Care* 1994;39(1):30-33.
17. Kaye W, Mancini ME. Retention of cardiopulmonary resuscitation skills by physicians, registered nurses, and the general public. *Crit Care Med* 1986;14(7):620-2.
18. Curry L, Gass D. Effects of training in cardiopulmonary resuscitation

- tion on competence and patient outcome. *Can Med Assoc J* 1987;137(6):491-496.
19. Yakei ME. Retention of cardiopulmonary resuscitation skills among nursing personnel: what makes the difference? *Heart Lung* 1989;18(5):520-5.
20. Wynne G, Marteau T, Evans TR. Instructors—a weak link in resuscitation training. *J R Coll Physicians Lond* 1992;26(4):372-3.
21. Kaye W, Rallis SF, Mancini ME, Linhares KC, Angell ML, Donovan DS, Zajano NC, Finger JA. The problem of poor retention of cardiopulmonary resuscitation skills may lie with the instructor, not the learner or the curriculum. *Resuscitation* 1991;21(1):67-87.
22. Moser DK, Coleman S. Recommendations for improving cardiopulmonary resuscitation skills retention. *Heart Lung* 1992;21(4):372-380.
23. American Heart Association. *Instructor's Manual for Advanced Cardiac Life Support*. Dallas: American Heart Association, 1994:5-4.
24. Billi JE, Membrino GE. Education in adult advanced cardiac life support training programs: changing the paradigm. *Ann Emerg Med* 1993 Feb;22(2 Pt 2):475-83.
25. Stross JK. Maintaining competency in advanced cardiac life support skills. *JAMA* 1983;249(24):3339-41.
26. Actronics, Inc. *The CPR/ACLS Multimedia Courseware* (1994 product literature). Pittsburgh: Actronics, Inc.
27. Jones A. Interactive videodisc technology: An overview for respiratory care education. *Respir Care* 1989;34(10):890-901.
28. American Heart Association. *Instructor's Manual for Advanced Cardiac Life Support*. Dallas: American Heart Association, 1994:B-5.
29. Rubens AJ. Testing Airway Management Skills: Interactive Video Courseware vs ACLS Instructor. *Respir Care* 1991;36(8):849-856.
30. Umlauf MG. How to provide around-the-clock CPR certification without losing any sleep. *J Contin Educ Nurs* 1990;21(6):248-253.
31. Rubens AJ. Effectiveness of an interactive videodisc system to teach the cognitive and psychomotor skills of airway management (doctoral dissertation). Pittsburgh PA: University of Pittsburgh, 1990.
32. Tanner TB. A computer simulation of cardiac emergencies. *Proc Annu Symp Comput Appl Med Care* 1991;894-896.
33. Wormuth DW. An innovative method of teaching Advanced Cardiac-Life Support, or why Cosmic Osmo may someday save your life. *Proc Annu Symp Comput Appl Med Care* 1991;864-865.
34. Mancini ME, Kaye W. Educational issues in basic and advanced cardiac life support. *J Cardiovasc Nurs* 1988;3(1):68-76.
35. Kaye W, Linhares KC, Breault RV, Norris PA, Stamoulis, CC, Khan AH. The mega code for training the advanced cardiac life support team. *Heart Lung* 1981;10(5):860-865.
36. Kaye W, Mancini ME. Use of the mega code to evaluate team leader performance during advanced cardiac life support. *Crit Care Med* 1986;14(2):99-104.
37. Palmisano JM, Akingbola OA, Moler FW, Custer JR. Simulated pediatric cardiopulmonary resuscitation: Initial events and response times of a hospital arrest team. *Respir Care* 1994;39(7):725-729.
38. Sullivan MJ, Guyatt GH. Simulated cardiac arrests for monitoring quality of in-hospital resuscitation. *Lancet* 1986;2(8507):618-620.
39. Eliadi C, Barbone M, Cyr S, Heard S. Monitoring of resuscitation performance standards using a mock code program (abstract). *Crit Care Med* 1992;20:834.
40. Funkhouser MJ, Hayward MF. Multidisciplinary mock codes: dream it, plan it, do it, rate it. *J Nurs Staff Dev* 1989;5:231-237.
41. Papadopoulos DA. Cardiopulmonary resuscitation training in a family medicine residency. *J Fam Pract* 1981;12(6):1013-1016.
42. Perlson R. Safe and successful delivery-room intubation and resuscitation of meconium-stained newborns by respiratory therapists. *Respir Care* 1986;31(8):689-694.
43. McLaughlin AJ Jr, Scott W. Training and evaluation of respiratory therapists in emergency intubation. *Respir Care* 1981;26(4):333-335.
44. Bryan HJ, Andree RA. Training respiratory therapists in emergency endotracheal intubation. *Respir Care* 1971;16:157-158. (Reprinted in *Respir Care* 1990;35(12):1206-1207.
45. Buck C, Southern PA. Who should perform endotracheal intubation? A summary of issues (editorial). *Respir Care* 1994;39(1):17-18.
46. Wood RY. Use of the nursing simulation laboratory in reentry programs: An innovative setting for updating clinical skills. *J Contin Educ Nurs* 1994;25(1):28-31.
47. Nelson MS, Traub S. Clinical skills training of U.S. medical students. *Acad Med* 1993;68(12):926-8.
48. Barrows HS. An overview of the uses of standardized patients for teaching and evaluating clinical skills. *Proceedings of the AACM's Consensus Conference*. *Acad Med* 1993;68(6):443-51.
49. Anderson MN, Kassebaum DG, editors. *Proceedings of the AACM's consensus conference on the use of standardized patients in the teaching and evaluation of clinical skills*. *Acad Med* 1993;68(6):437-453.
50. Vu NV, Barrows HS, Marcy ML, Verhulst SJ, Collier JA, Travis T. Six years of comprehensive, clinical, performance-based assessment using standardized patients at the Southern Illinois University School of Medicine. *Acad Med* 1992;67(1):42-50.
51. Morhaim DK, Heller MB. The practice of teaching endotracheal intubation on recently deceased patients. *J Emerg Med* 1991;9(6):515-518.
52. Iserson KV. Postmortem procedures in the emergency department: using the recently dead to practice and teach. *J Med Ethics* 1993;19(2):92-98.
53. Benfield DG, Flaksman RJ, Lin TH, Kantak AD, Kokomoor FW, Vollman JH. Teaching intubation skills using newly deceased infants. *JAMA* 1991;265(18):2360-2363.
54. American Heart Association: *Textbook for Advance Cardiac Life Support*. Dallas: American Heart Association, 1994:15-5.
55. Nelson MS. Models for teaching emergency medicine skills. *Ann Emerg Med* 1990;19(3):333-335.
56. Kislung JA, Schreiner RL. The kitten as a teaching model for intubation of the human neonate. *Respir Care* 1976;21:1243-1246.
57. Burns T. Emergency Care and ACLS (letter). *Respir Care* 1992;37(8):947-948.
58. Horn C. ACLS skills for the respiratory therapist (letter). *Respir Care* 1992;37(8):945.
59. Rinaldo Gallo S. More on ACLS training (letter). *Respir Care* 1992;37(8):945-946.
60. Shaffer T. In support of ACLS certification (letter). *Respir Care* 1992;37(12):1446-1447.
61. Demers B. My Parting Shot (president's perspective). *AARC Times* 1992;16(12):6-7.
62. Kaye W, Mancini ME, Rallis SF, Linhares KC, Angell ML, Donovan DS, Zajano NC, Finger JA Jr. Can better basic and advanced cardiac life support improve outcome from cardiac arrest? *Crit Care Med* 1985;13(11):916-20.
63. Donofrio MT, Barron DM, Winters SL, Gomes JA, Gorlin R. Cardiopulmonary resuscitations: qualifications and performance of house staff and trained internists. *Mt Sinai J Med* 1989;56:279-284.
64. Birnbaum ML, Kuska BM, Stone HL, Robinson NE. Need for ad-

vanced cardiac life-support training in rural, community hospitals. *Crit Care Med* 1994;22(5):735-740.

65. Sanders AB, Berg RA, Bures M, Genova RT, Kern KB, Ewy GA. The efficacy of an ACLS training program for resuscitation from cardiac arrest in a rural community. *Ann Emerg Med* 1994;23(1):56-9.
66. American Heart Association. Instructor's Manual for Advanced Cardiac Life Support. Dallas: American Heart Association, 1994:5-2.
67. Standards and Guidelines for Cardiopulmonary Resuscitation (CPR) and Emergency Cardiac Care (ECC). *JAMA* 1980;244:353-509.
68. Billi JE. The educational direction of the ACLS training program.

Ann Emerg Med 1993 Feb;22(2 Pt 2):484-488.

69. American Heart Association. Instructor's Manual for Advanced Cardiac Life Support. Dallas: American Heart Association, 1994:2-3.
70. Joint Commission on the Accreditation of Healthcare Organizations. Accreditation manual for hospitals (Volume 1: standards, Section 4: Orientation, training, and education of staff). Oakbrook Terrace IL: JCAHO, 1994:149-150.
71. American Heart Association. Instructor's Manual for Advanced Cardiac Life Support. Dallas: American Heart Association, 1994:C17-2.
72. American Heart Association: Textbook for Advance Cardiac Life Support. Dallas: American Heart Association, 1994:15-7.

Boudin Discussion

Durbin: The American Heart Association has removed the requirement that physicians act as course directors. Each of the states has to decide what the standard in that state should be. Most affiliates, I think, will go along with the AHA recommendation. This should make a huge difference in the ability to put on courses for various and sundry groups, and, I think, will help the process along immensely even though it may take a year or two to get going. The new administrative manual should help a lot. May I ask, did you see any reports on videotaping codes and debriefing? Could this be used as a criterion for recertification or evaluation of performance?

Boudin: Videotaping mock codes or simulated cardiac arrests as a method of evaluating performance has been reported in the literature.¹ An audio-cassette tape and written record has also been shown to provide useful information.²

1. Papadopoulos DA. Cardiopulmonary resuscitation training in a family medicine residency. *J Fam Practice* 1981;12(6):1013-1016.
2. Palmisano JM, Akingbola OA, Moler FW, Custer JR. Simulated pediatric cardiopulmonary resuscitation: initial events and response times of a hospital arrest team. *Respir Care* 1994;39(7):725-729.

Mathews: Karen, I have 2 comments,

one regarding videotaping. We used to do that when I was at Yale a long time ago, until the Legal Counsel's Office found out about it. Then they put a quick stop to it. We used to have cameras on all the beds in one ICU, but the lawyers just said Absolutely not! So, if you're going to use videotapes as a training aid, you'd better use simulated patients, not real ones. Secondly, I was a little concerned that, when you discussed the interactive videos computer simulations, you didn't point out that this is not really skills training, but cognitive testing. You can't teach people psychomotor skills using the interactive videos and the like, and many of the skills we're talking about are psychomotor skills. We have to take that into consideration, too. I would suggest that a nice combination would be Dr Kaye's Megacode with the computer simulation for the cognitive and maybe some of the affective domain.

Boudin: Actually, interactive videodisc technology is not limited to cognitive training. You can use the computer system for teaching psychomotor skills when you have the appropriate mannequins that interact with the computer. The information, supplied by Actronics Inc¹ states that use of their courseware and test station is a valid method of obtaining course completion in BLS and ACLS.

1. Actronics Inc. The CPR/ACLS multimedia courseware (product literature). Pittsburgh: Actronics Inc, 1994.

Mathews: That may well be true for cognitive skills training, but I don't see how you can do actual procedures. You can say, "Yes, this is the correct procedure to use," but you can't do that. You can't do cardiac massage or intubation using that technology. It just isn't possible at this time.

Barnes: Art (Sanders) probably wants to comment on this too, but I was just talking with the Actronics people at the last Heart Association meeting, and part of the great expense is that they have sensors built into a Laerdal mannequin, and I think the mannequin with the sensor system is about \$22,000. I think it does go quite a long way toward measuring the psychomotor skills using these sensors.

Sanders: That's exactly right. The technology is available to teach and evaluate psychomotor skills and knowledge. Sophisticated mannequins can teach and assess CPR as well as endotracheal intubation. Some adults learn well using these devices and pace themselves and their progress. Others find it harder to reach course completion on the mannequin because it requires perfect CPR.

Barnes: Pat Brougner sent me a very interesting paper recently describing the videotaping of difficult intubations in the emergency room. It was a very elaborate system, not only to videotape the actual procedure, but all the data were recorded on videotape,

sound was recorded. They found it very useful for difficult intubations that were done incorrectly when they had a debriefing session afterward because the people involved couldn't remember the specifics of what happened. When they watched the videotape, listened to the audiotapes, and actually reviewed the data recorded simultaneously, they were amazed at how much they had forgotten and how things had changed from the anecdotal versions that they had gotten second or third hand. So, there's been quite a bit of work, and I think a lot of us have read the experience with the emergency trauma room where they videotaped the trauma team over a period of 6 months. Each week they watched the videotapes and the performance of the trauma team. They were able to show that the performance of the trauma team actually improved over 6 months because of their tapes.

Masferrer: I don't understand why all RCPs are not ACLS-trained and why the schools of respiratory therapy don't train people in ACLS by the time they graduate.

Boudin: I know that Bob Fluck is going to talk about the training of students in his presentation, which is next. Why don't all respiratory care practitioners take ACLS? Well, for one, I believe that, in the hospitals, it hasn't been emphasized as an important skill for RCPs. There's also the feeling that may be expressed, Why should I learn ACLS when I don't do ACLS? I'd like to turn the question around to them and ask, How can you expect to perform ACLS if you refuse to learn the skills? The first steps are to adopt a professional attitude and to want to learn the necessary information.

Masferrer: Let's say that I want to become ACLS-trained when I go back to Dallas.

Boudin: It's really not as difficult as many people believe it to be. With adequate preparation and encouragement, it's very easily accomplished.

Barnes: Let me make an attempt at this. I've been wrestling with this, as you know, for a long time. It's extremely frustrating. I would say that the good schools do provide ACLS-provider training right before the student graduates and the poor schools don't. I can tell you that there are a lot of 1-year schools that probably shouldn't exist in our field. The technology has gone far enough that you really can't train an entry-level person in 1 year or 15 months. We need a minimum associate degree entry-level in our field, like the AS degree nurse and the BSN nurse. That's long overdue, and the fact of the matter is we have 180 1-year technician programs. People in hospitals don't know the difference between a registered therapist and a certified respiratory therapy technician. That's not to say that some individual technicians can't function at a therapist level through on-the-job training or individual initiative. What we need is a common across-the-board standard with pharmacology, time for discussions on intravenous infusions, and all the things that we're hoping therapists will do. Students really can't learn mechanical ventilation, acid-base balance and the rest of what we give them and fit ACLS into a 15-month or 12-month curriculum. Just one last comment. We work all over the hospital. When we talk to young people about careers in respiratory care, ACLS is one of the things we emphasize. The excitement of the field is that you'll be working with a neonate at one moment and a geriatric patient the next, a surgical patient in one part of the day and an emergency room patient in another part. We literally travel the hospital. So when things happen, we're there. That means that we just can't have an

elite group who are highly trained. The average therapist who is all over the hospital has to have a minimum base of ACLS skills. So, I'm very much in favor of having everybody have ACLS in school. And, as Ray implied, that's really an indictment of the schools. There should be a lot of embarrassed program directors.

Rau: I'm a program director, so I'll be embarrassed. I'd like to put it in a different perspective if I could, though. Tom, I think you sort of digressed a little bit and used that as a platform to talk about getting rid of 1-year programs, and certainly, it's a big jump there. If we require ACLS, that would be a big argument for getting rid of 1-year programs, but I think that's a separate issue, because the first question, the logically prior question, is ACLS. We can train, and we do have an ACLS course in our curriculum. If you are training them for skills that they are not able to practice in the workplace, you create a frustrated student. I was frustrated as a student—a long time ago, back in 1972 when I got into the field—because I learned things that I couldn't practice. In some places we don't really even manage ventilators. We are told which knob to turn and what number to put it on or which part of the membrane touch pad to hit. I don't think it's the school's issue, and my response to you would be that we have to put it into the perspective by saying, This is the workplace. We've got to synchronize with the schools. We can pass an ordinance and say every school should have an ACLS course, but it's got to happen in the workplace, and then we can satisfy the workplace demand.

Barnes: You cannot expect medical directors and medical committees to allow RCPs to exercise these skills unless they have the education first. I think that education has to come first. There may be a really tough period of

time—2-5 years—where people are chafing at the bit because they're highly educated and trained and they can't do these things. But that has to come first. It's one of the first questions licensure boards ask. What was the education and the training related to this particular skill?

Malinowski: As a former educator in a respiratory care program, I can tell you that two things that dictate our curriculum are the job market, what employers tell us they need, and the NBRC test matrix. Many of us are held to evaluation on these two aspects because they are integral to accreditation. When evaluating the outcomes of your program, you must look at how many of your students pass the boards. The NBRC matrix is based on job analyses that do not show that respiratory therapists start I.V.s, administer drugs, or perform many other ACLS skills. When you're trying to fit all the information that's required into a very short curriculum, it's very difficult. Our baccalaureate-degree program incorporates ACLS training, but it's very difficult to fit it into the associate-degree program. Our employers are now telling us that they want therapists who can do electrocardiograms and who are skilled in many other areas. Some hospitals are training RCPs to wash patients and empty bedpans. Our educational system is having difficulty producing a student who can meet the changing needs of health care at a cost it is willing to pay and in the time allotted. So, while I personally think ACLS training is valuable, again, if it's not being requested by employers or required by the creden-

tialing board, it is difficult for educators to justify providing it.

Boudin: The diagram I showed was actually a random sample of only 8 hospitals I surveyed. In no way was it meant to be inclusive of all the hospitals that train RCPs in ACLS. It would be difficult to conduct a survey of all the hospitals in the United States, but, certainly, such information would be interesting.

I do want to make a point about the advantage an ACLS-trained RCP has in the job market—Respiratory care department administrators can be very impressed by seeing this on an applicant's résumé. They move that application to the top of the stack, even if ACLS is not a requirement for their department, and that applicant has a better opportunity of getting a position when there are a limited number of openings available. It really speaks for professionalism, just as the RRT credential and the other NBRC credentials do.

Fluck: The RRT exam matrix does include sections on defibrillation, administration of epinephrine and bicarbonate, airway management—all ACLS skills. So they are already there, which means employers are already looking for people to do those kinds of things.

Malinowski: But that information is taught in typical curricula. If you look at ACLS skills, therapists have a lot of them already. Maybe the new format—an educational program instead of a certification—will facilitate the incorporation of knowledge and skills already in place in the RCP's educa-

tional program. My concern is that just adding additional time, practice, testing, and study to programs that are already struggling to achieve what is necessary for the advanced skills and knowledge now being demanded of their graduates is difficult.

Fluck: We just have to decide that it's a priority, and do it.

Kaye: Be careful, however, because I think that you're still going to have to devote a lot of precourse emphasis on those areas where RCPs may have no expertise—for example, arrhythmias and drugs. Ultimately, it must be in the schools. I agree that that's the answer. But I don't think that the situation is any different for medical students or even junior critical care nurses. I think it's a little hill, not a big hill. A couple of other comments. Karen, I hope that the people with whom you've been in touch who have done these ACLS courses are going to publish their results. It's terribly important, and I think maybe one of the roles you can play is get to them and say, Please publish your results—especially if they're using them for their quality improvement program.

Boudin: I'll do that, Bill. Most of the respiratory care departments that I contacted were very enthusiastic to provide information when I told them their experiences were going to be published in *RESPIRATORY CARE*.

Kaye: But encourage them to evaluate performance. We need some post-training data to show that therapists can learn just as well as doctors and nurses. It's no different.

The First ACLS Course—Resuscitation Training for Students

Robert R Fluck Jr MS EMT-P RRT

- I. Introduction
- II. Why Should Respiratory Care Practitioners Learn ACLS?
- III. Why Should Respiratory Care Students Learn ACLS?
- IV. What Skills Should Students Master?
- V. How Should Competency Be Evaluated?
- VI. Can Students Learn All of the Material during the Course?
- VII. What Material Needs To Be Taught in Anticipation?
- VIII. Where in the Curriculum Should the Course Be Scheduled?
- IX. Should Instructors Other Than Program Faculty Be Used?
- X. What Psychological Preparation of Students Is Needed?
- XI. Is Apprenticeship with a Senior Practitioner Useful?
- XII. In Conclusion

Introduction

Before addressing the issue of Advanced Cardiac Life Support (ACLS) training for students, it is best to organize one's thoughts by deciding the key questions that need to be answered. It may also be useful to note the distinction between the word education and the word training because we chose training for the title of this article. Both terms connote conveying information and experience. The aim in education is to improve the person's knowledge, in training, the person's skills or performance.¹ In spite of the pejorative connotation of training (eg, "We could train a monkey to do that!"), it is a useful distinction. In an ACLS course, participants need to do things during the course, not merely absorb knowledge like sponges. Although the term training has received undeserved 'bad press,' emphasis on education without training in any endeavor that involves both knowledge and psychomotor performance is wholly

inappropriate. The reader is directed to the Clinical Practice Guidelines published by the American Association for Respiratory Care; all of them (for example, *Resuscitation in Acute Care Hospitals*²) emphasize both knowledge and demonstrated ability to perform.

ACLS courses have been taught for more than 20 years—since the first course was developed by the Nebraska Affiliate of the American Heart Association (AHA).³ However, it has been only in the last 5 years that we have seen a greater effort exerted to have respiratory care practitioners (RCPs) participate in ACLS training.

Why Should Respiratory Care Practitioners Learn ACLS?

In almost all hospital situations, the RCP has flexibility and a knowledge of the entire hospital that make him or her valuable anywhere in house as a member of the code, or resuscitation, team. Further, with few exceptions, respiratory care personnel are in the hospital 24 hours a day, 7 days a week, throughout the year. In smaller community hospitals, the RCP may well be the only person present in the hospital who has knowledge and training in airway management, positive pressure ventilation (both manual and mechanical), and blood-gas sampling, analysis, and evaluation.

Next, the AARC Clinical Practice Guideline—*Resuscitation in Acute Care Hospitals*—specifies that Level-II personnel be trained, evaluated, and retrained in ACLS, at intervals of no longer than 1 year.² The recom-

Mr Fluck is Associate Professor and Clinical Coordinator, Department of Respiratory Care and Cardiorespiratory Sciences, SUNY-Health Science Center, Syracuse, New York.

A version of this paper was presented by Mr Fluck during the RESPIRATORY CARE Journal Conference "Resuscitation in Acute Care Hospitals" held in Cancun, Mexico, October 21-23, 1994.

Reprints: Robert R Fluck Jr, Department of Respiratory Care and Cardiorespiratory Sciences, SUNY-Health Science Center, 750 E Adams, Syracuse NY 13210.

mentation for a 1-year retraining interval deserves mention because the AHA-recommended interval is 2 years (as entered on course-completion cards). In 1983, Stross⁴ discovered that only slightly more than 50% of physicians trained in ACLS would initiate appropriate therapy when presented a mock arrest scenario 1 year following their training. He, therefore, recommended yearly recertification.

In 1992, Barnes and Durbin⁵ recommended that respiratory therapists who frequently assist with resuscitations receive ACLS training. Two members of the AHA's ACLS Education Panel have said, "The sole purpose of the ACLS Training Program is the education of health professionals whose jobs include the management of arrest and near-arrest patients."⁶ In September 1992, then-AARC President Bob Demers asked for membership input into a proposed position statement that would recommend ACLS training for all respiratory care practitioners practicing in critical care units and emergency departments.⁷ Several practitioners wrote to provide support for the notion of RCPs' being trained in ACLS. One therapist in home care⁸ wrote to encourage immediate implementation of the Barnes and Durbin recommendation. Reasons that he gave for immediate implementation included improving credibility, enhancing knowledge and skills, and increased earning power as a result of the increased utility of the practitioners. Bearden, an ACLS coordinator⁹ mentioned her department's coordination of ACLS classes for her hospital and the accessibility that this affords RCPs to the training. She included respect from other health-care providers and citation in the RCP's yearly performance evaluation as benefits. Gallo¹⁰ agreed with Barnes and Durbin and pointed out that having RCPs who are ACLS instructors in her department makes it more economical to achieve her department's goal of having all RCPs who perform endotracheal intubation achieve ACLS certification. Henson¹¹ expressed surprise that more departments have not achieved what his department had achieved 1 year earlier—having all staff complete ACLS training. The major benefit he mentions is increased professionalism among department staff, facilitating their dealing with other health-care professionals on an equal basis. His staff speak up during resuscitations when they feel something can be improved.

In the second edition of the ACLS textbook,¹² the AHA emphasizes two objectives of ACLS training: (1) to disseminate the guidelines as broadly as possible; and (2) to encourage the interdisciplinary nature of the ACLS program. Although RCPs are not specially mentioned, both of these goals, make it reasonable for RCPs to be included in ACLS training on an equal footing with other health-care workers.

Donofrio and colleagues¹³ found, among house officers who had ACLS training, increased confidence in their ability to resuscitate appropriately. Funkhouser and Hayward¹⁴ found better performance, more confidence, and less anxiety

during simulated resuscitation exercises among those who had completed ACLS training.

In evaluating the knowledge level of practitioners in 12 rural hospitals, Birnbaum and associates¹⁵ found "substantial" weakness in electrocardiogram (ECG) interpretation, interpretation of acid-base disturbances, pharmacology, and airway management techniques. It is important to note that RCPs comprised only 9 of 461 people who were evaluated for this study. However, RCPs are experts in airway management and acid-base interpretation and receive education and training in pharmacology related to resuscitation and in ECG interpretation. Thus, ACLS training can only enhance their utility during resuscitations in these small hospitals.

In spite of the many benefits and advantages of ACLS training for RCPs, all is not well everywhere. Burns¹⁶ wrote in a letter to this journal of a "backlash" against active therapist participation in resuscitation, specifically related to performing intubations. He cites many "less than sincere" reasons expressed by those advocating that only physicians intubate. There is what I consider to be a less-than-subtle derogation of RCPs contained in the latest ACLS Instructor's Manual.¹⁷ Under the section titled "Problems in ACLS Education: More Tricks of the Trade," a scenario is presented in which two of the students in an ACLS Instructor's Course are respiratory therapists. The students are described as being "uninterested, holding back, not getting involved" and having "only the most cursory understanding of ACLS concepts." No other health profession is singled out as being a potential problem in ACLS training.

Why Should Respiratory Care Students Learn ACLS?

Barnes and Durbin⁵ assert that it makes the student (as a graduate) more "marketable," a sentiment echoed by Hunter, as quoted in a recent article.¹⁸ They reiterate the points made by Donofrio et al¹³ and Funkhouser and Hayward¹⁴ that those with ACLS training are more confident, perform more competently, and have less anxiety during resuscitation situations. Shaffer¹⁹ has offered ACLS as an option to her students for 7 years; more than 80% now take the course. She believes that the benefits accruing from ACLS training for students include a higher level of credibility and improved interdisciplinary relationships.

What Skills Should Students Master?

We turn our attention next to those skills that students should master. Although the answer may seem obvious to the most casual observer, it may not be. For example, ACLS training in our institution was recently moved from

Critical Care to Emergency Medicine, and, as a consequence, medical directors were changed. I called to introduce myself to the new medical director, and we talked for a few minutes. In the course of the conversation, he asked me whether we delivered an ACLS course tailored to respiratory therapists and what skills we expected them to master. I was taken aback by this, but quickly responded that we expected them to master *all* of the required skills. (In fact, we spend more time on non-RCP skills, such as I.V. insertion, specifically because they have not been previously trained in those.) The medical director's response seems to reflect a mindset of compartmentalizing therapists' knowledge and skills as being limited only to airway management. However, because most of the hospital beds in the United States are in smaller community hospitals, it makes *more* sense to have therapists skilled in all aspects of resuscitation. In community hospitals, no physicians may be present most of the time. When a resuscitation does occur, nurses and respiratory care practitioners perform the resuscitation. There is no reason that the RCP cannot be the team leader or function in *any* position on the team. Further, in larger hospitals with house staff, the therapist may arrive before the house staff; because time is crucial in resuscitation, it is important that the RCPs be trained to take possibly life-saving or life-sustaining actions prior to the arrival of the house officer—if the hospital allows him to do so.

How Should Competency Be Evaluated?

Evaluation of competency related to ACLS has undergone important changes with the publishing of the new Instructor's Manual.¹⁷ Under the old course format (with the exception of the much-feared Megacode station), skills and knowledge were evaluated at stations that merely tested psychomotor skills (and perhaps some knowledge) 'in a vacuum.' There was a station at which one started an I.V. line; one at which one inserted airways and endotracheal tubes; one at which one interpreted 'static' EKG rhythm strips; another at which one interpreted the rhythm from a monitor screen and defibrillated as appropriate. Only at the Megacode station did the student actually apply what he was learning in a simulation—interpret an ECG rhythm, determine and deliver appropriate therapy, evaluate the patient's response to the therapy, and alter therapy as appropriate. This paralleled the way in which the course was taught. There was a pharmacology lecture, an airway-management and oxygen-therapy lecture, an EKG-interpretation lecture, a lecture on special resuscitation situations, even (incredibly enough) a lecture on "putting it all together" (application).

Most of the students in an ACLS course are adults older than 20 years of age. The teaching/learning methods listed

violate several of the tenets of adult education. Most importantly, the students need to be able to apply (use) immediately what they have learned. They also need to see the relevance of what they are learning. Finally, they need to build upon and connect these new skills and knowledge to what they already know and can do. The new ACLS Instructor's Manual uses entirely scenario-based learning and evaluating. The students are evaluated as they practice in a given situation (such as a patient in stable supraventricular tachycardia). All the skills and knowledge necessary for successful treatment of this type of rhythm are learned in the context of the patient situation, with the patient giving a variable response to treatment and exhibiting the consequences of inappropriate treatment. If a student displays mastery during the teaching session, he does not have to return to be formally checked off at a testing session.

In fact, "mastery" may be defined differently for different people, even within the same course. Billi²⁰ makes this point in two different ways. Because rescuers may be called upon to perform different functions during an arrest situation, the level of perfection to which the person is held depends on the person's profession, role, and the practice setting. Secondly, the object of an ACLS course is to raise the level of knowledge and skills of each participant as much as possible, within the time limits of the course; each person need not necessarily achieve parity of skills and knowledge with all other course participants.

Can Students Learn All the Material during the Course Itself?

The answer seems self-evident—'certainly not.' Birnbaum and colleagues²¹ found that most of those who dropped out of their ACLS course were not adequately prepared ahead of time. (One can assume that these were hospital employees who had already graduated from health profession educational programs). The time format of an ACLS provider course is 3 days (a total of about 24 class hours) for neophytes, those who have never taken the course before or who do not use the course material on a frequent basis.

The volume of material covered during the course precludes effective mastery of the subjects unless the material has been introduced earlier. ECG interpretation alone can take several weeks and much practice to achieve even a minimum level of competency. The list of drugs, including actions, doses, contraindications, and side effects, is long and daunting to someone who has not seen it before. Finally, the different algorithms (especially the complex tachycardia algorithm) cannot possibly be learned during the short time of the actual course. Consequently, the pressure on students is high and may hinder the learning process. An alternative is proposed by King and Lee,²² who delivered the ACLS course for medical students over a 6-

day period, including a day off after 3 days. Their aggregate pass rate for all stations was 86% on the first attempt and 100% on the second.

What Material Needs To Be Taught in Anticipation?

If students cannot learn everything within the confines of the course, what needs to be taught in advance? ECG interpretation fits nicely into a cardiovascular physiology course; in fact, it is hard to imagine such a course without a unit on ECGs. Pharmacology also fits well into cardiovascular physiology because nearly all of the drugs affect the cardiovascular system directly. A general pharmacology course may also address such drugs; however, because of the number of drugs covered in a general course, ACLS drugs might not receive sufficient emphasis. General pharmacology would certainly be helpful as a basis, but the ACLS emphasis would need to be provided in the cardiovascular physiology course.

As an adjunct to simple lectures on subjects that need to be applied, there is at least one computerized simulation for resuscitation called *Cardiac Arrest!* (Mad Scientist Software, Alpine UT). It includes procedures, such as intubation, starting an I.V., hyperventilation, delivery of drugs by both bolus and infusion, some laboratory procedures (ABGs, electrolytes, glucose), and history. The learner can choose from among 45 different patients, from infant to adult and from easy-to-difficult resuscitations. The accompanying manual contains useful information about resuscitation beyond explaining how the program works. Even if a resuscitation is immediately repeated, the same patient does not respond in the same way to the interventions.

Some topics are naturally covered prior to an ACLS course, which is usually delivered late in the respiratory care curriculum. Oxygen therapy and airway management, although important, need less time in the ACLS course because they are emphasized in the respiratory care curriculum. Venipuncture technique is similar to arterial puncture. Although students may never have actually punctured a vein, the techniques are somewhat parallel.

Where in the Curriculum Should the Course Be Scheduled?

An ACLS course should be scheduled near the end of the respiratory care curriculum. Although the course stresses *application*, students need a base of *knowledge* in order to be successful. Our course is scheduled early in the last semester of a 2-year entry level therapist program that awards both associate and baccalaureate degrees (Fig. 1.) This places it close to the cardiovascular physiology course that the students finish during the previous

semester. It also follows nearly every other professional course: the two clinical laboratory courses, Medical Physics, Oxygen and Aerosol Therapy, and Respiratory Physiology, Prolonged Artificial Ventilation, Respiratory Care for the Medical-Surgical Patient (a disease course), and Neonatal-Pediatric Respiratory Care. Of the major professional courses, only Acid-Base Physiology remains, and the students have already received an introduction to this body of knowledge.

Fall Semester 1 Introduction to Respiratory Care (Clinical) Medical Physics Oxygen and Aerosol Therapy Physiology I Anatomy Expository Writing Clinical Lab	Spring Semester 1 Applied Respiratory Care I (Clinical) Cardiopulmonary Health and Disease Respiratory Physiology Prolonged Artificial Ventilation Physiology II Anatomy Clinical Lab
Summer Semester Summer Clerkship (Clinical) Neonatal-Pediatric Respiratory Care	
Fall Semester 2 Applied Respiratory Care II Cardiovascular Physiology Respiratory Care for the Medical-Surgical Patient General Chemistry (laboratory course) Pathology Introduction to Sociology	Spring Semester 2 Applied Respiratory Care III Acid-Base Physiology Respiratory Care Administration Biological Chemistry (with laboratory) Pharmacology Introduction to Psychology Introduction to Literature Home Care and Rehabilitation

Fig. 1. Program of study for the Associate of Arts degree at SUNY-Health Science Center at Syracuse. Baccalaureate degree students take their professional courses in the same order.

Clinical simulation exams are used extensively in our program to provide practice at the application level. Our students have had problems in the past with the parts of the National Board for Respiratory Care Written Registry Examination dealing with patient evaluation and recommendations for changes in therapy and also with the Clinical Simulation Examination. We have increased our use of simulations (with instructor guidance) to improve performance on these aspects of application. Without some background in assessing the patient and applying knowl-

edge, it seems unlikely that a respiratory care student would meet with success in the ACLS provider course. Hence, scheduling the course late in the respiratory care curriculum appears to make sense.

Should Instructors Other Than Program Faculty Be Used?

Using instructors other than the faculty of the department for teaching the course makes sense in order to obtain an unbiased third-party appraisal of the students' abilities. We formerly used physicians from the Departments of Emergency Medicine and Critical Care Medicine to evaluate the Megacode station, and their evaluation gave us valuable feedback. Because the key scenario in the new ACLS course is ventricular fibrillation/pulseless ventricular tachycardia, we plan to use physicians to teach and evaluate that scenario after the students have been through it at least once. Regardless of where they are ultimately used, physicians must be a part of the course faculty to help us evaluate student performance. In a content area such as this, it is important to validate the training process.

What Psychological Preparation of Students Is Needed?

The need for psychological preparation of students, especially with the new ACLS training format, will be lessened. With the earlier format there was pressure to pass the written exam and all of the stations. That pressure was evident during the course and especially during the testing days. Birnbaum, et al²¹ concluded that most personnel whose duties included participation in resuscitations could achieve the objectives of the course. The psychological preparation should include early emphasis on establishing a sound base of knowledge. When we first began ACLS training for our students, one student complained about having ACLS topics covered during another course. Students need to understand that higher level objectives (at the application level) need more time to fulfill and depend upon thorough knowledge of the subject. Although students should not receive the impression that ACLS training is easy, their fears of the written exam and the Megacode station can be assuaged.

Is Apprenticeship with a Senior Practitioner Useful?

The importance of mentoring has been pointed out.²³ The immediate environment of a resuscitation can be as stressful a place as one can find in a hospital. It is unreasonable to expect someone who has only watched a code to function immediately on his own, as a member of the team.

More than simply being useful, mentoring allows the neophyte practitioner to easily and with a minimum of stress become a fully functioning member of the resuscitation team. By first modeling and then helping the new practitioner perform his duties during the resuscitation, the mentor facilitates the learning and transition processes.

In Conclusion

Learning and applying the skills associated with ACLS are scary at best. When one is performing BLS (especially in a hospital), there is always the knowledge that someone will be doing ACLS, and although effective BLS is necessary for a successful outcome, proper ACLS is essential. For the student who has never been part of a resuscitation team, initially assuming the role of an ACLS provider can be doubly frightening. By laying the proper foundation for the ACLS course, conducting the course in an effective and nonthreatening manner, and providing for a formal protegee-mentor relationship during the new graduate's orientation, I believe that we can assure that each respiratory care practitioner reaches his maximal potential as a member of the resuscitation team.

REFERENCES

1. Howlett H. Why training doesn't work and what to do about it. Association for Quality and Participation Newsletter, 1994; August/September:10.
2. American Association for Respiratory Care. Clinical Practice Guideline Resuscitation in Acute Care Hospitals. *Respir Care* 1993; 38:1179-1188.
3. American Heart Association. Instructor's Manual for Advanced Cardiac Life Support. Dallas; American Heart Association, 1988.
4. Stross JK. Maintaining competence in Advanced Cardiac Life Support skills. *JAMA* 1983; 249:3339-3341.
5. Barnes TA, Durbin CG Jr. ACLS skills for the respiratory therapist: time for a mandate. *Respir Care* 1992; 37:516-519.
6. Billi JE, Membrino GE. Education in adult advanced cardiac life support training programs: changing the paradigm. *Ann Emerg Med* 1993; 22:209-217.
7. Demers B. Mandatory ACLS training for RCPs: a referendum. *AARC Times* 1992;16:8-11.
8. Horn C. ACLS skills for the respiratory therapist (letter). *Respir Care* 1992;37:945.
9. Bearden EF. ACLS—One department's experience (letter). *Respir Care* 1992;37:945.
10. Gallo SR. More on ACLS training (letter). *Respir Care* 1992;37:945-946.
11. Henson D. Dinosaurs, RCPs, and ACLS (letter). *Respir Care* 1992;37:946-947.
12. American Heart Association. Textbook of Advanced Cardiac Life Support, 2nd ed. Dallas; American Heart Association, 1990.
13. Donofrio MT, Barron DM, Winters SL, Gomes JA, Gorlin R. Cardiopulmonary resuscitation: qualifications and performance of housestaff and trained internists. *Mt Sinai J Med* 1989;56:279-284.

14. Funkhouser MJ, Hayward MF. Multidisciplinary mock codes: dream it, plan it, do it, rate it. *J Nurs Staff Dev* 1989;5:231-237.
15. Birnbaum ML, Kuska BM, Stone HL, Robinson NE. Need for advanced cardiac life-support training in rural, community hospitals. *Crit Care Med* 1994;22:735-740.
16. Burns T. Emergency care and ACLS (letter). *Respir Care* 1992;37:947-948.
17. Billi J, Cummins RO, eds. *Instructor's manual for Advanced Cardiac Life Support*. Dallas: American Heart Association, 1994.
18. Gibbons M. Students get singular message—jobs aren't falling from trees this year. *Advance for Respiratory Care Practitioners* 1994 Aug 29;7:9.
19. Shaffer T. In support of ACLS certification (letter). *Respir Care* 1992; 37:1446-1447.
20. Billi JE. The educational direction of the ACLS training program. *Ann Emerg Med* 1993;22:218-222.
21. Birnbaum ML, Robinson NE, Kuska BM, Stone HL, Fryback DG, Rose JH. Effect of advanced cardiac life-support training in rural, community hospitals. *Crit Care Med* 1994;22:741-749.
22. King KP, Lee LS. ACLS education for medical students: A "distributive" course format (poster). *Anesthesiology* 1994;81:A1249.
23. Cullen DL, Luna G. Mentoring: a valuable tool for professional development. *AARCTimes* 1994;18:34-35,66.

Fluck Discussion

Barnes: The AARC was asked by the CPR Focus Group to come out with a national position paper saying that all respiratory care practitioners should be trained in ACLS. The request went to the Board of Directors of the AARC, then to AARC's Board of Medical Advisors, made up of several physicians from different organizations. The response of the Board of Medical Advisors to the Board of Directors was that they were not going to endorse a position paper until we can show the Board of Medical Advisors that the schools adequately prepare respiratory therapists to assume these skills. They wanted to see evidence that the things that Bob (Fluck) is talking about are being done very well in the schools first, before there is a national position paper.

Fluck: I think that's a really important point. This is the first I've heard of that. I saw Bob Demers's editorial in *AARC Times*¹ and that appeared to be the end of it.

1. Demers B. Mandatory ACLS training for RCPs: a referendum. *AARC Times* 1992;16(9):8,11.

Bishop: I think that the respiratory therapists here are all leaders, recognized leaders in the field, and at the forefront. Two years ago I came from being a medical director at a hospital with a very active respiratory therapy

department to one where we're really at a different level. I said, "Look, I really think that we need to have respiratory therapists as an integral part of the code team, and I think it would be a good idea if the people were intuiting and were ACLS certified." Quite frankly, we do not have the resources to do it nor could I generate a strong interest from our therapists for ACLS training.

Barnes: We have feedback from our local emergency cardiac care committee saying that ACLS courses are designed for practitioners, and that it's really inappropriate for students to take the course because they're not out practicing. But, I think student training is critical because when you give a graduating student an ACLS course, you're exposing him to the importance of the material with the hope that he will retrain every year or two. You're actually starting him down the right road, rather than just taking a chance that he will leave school and take the course a year or two later.

Bishop: I agree with you, you must start people down that road, but I'm not convinced that there is as much support among all the respiratory care departments as there is among you, the leaders in that field.

Fluck: I think role modeling has a place here again as well. If the director of the department is the role model and indicates that ACLS is the

standard for the department ... I think it has to come from the top.

Rodriguez: We have not mentioned that when respiratory care practitioners compete for funds for advanced training, especially in an era of cost reductions, we often find ourselves on the bottom rung. I think that if there is a perception that entry-level respiratory care practitioners are well prepared to function in the critical care area without additional training, hospital administrators will decide to expand their role in ACLS, with greater willingness and confidence. We really need to begin by introducing ACLS at the student level.

Fluck: It's not that big a deal either; three of our faculty are ACLS instructors. Now with the new Heart Association guidelines, although you still have to have a physician-instructor in your course, he does not have to be the course director. That makes it even easier.

Paul Mathews: We may have one of the longest experience with teaching ACLS in the respiratory care programs. We've been doing this for about 8 years now. We've trained over 100 people as ACLS providers—senior and baccalaureate students. We have a 100% pass rate: everyone of those students passes—they don't graduate from the program unless they have successfully completed the ACLS provider course. The reason we

think it's successful is that we spend a lot of time up front doing exactly what Bob (Fluck) said. We give them a lot of this material up front; we have a concentrated 6- to 8- week course that the students take—1 day a week (2 hours per day)—and they do really well. We have these courses all evaluated by outside people—the people who teach the course don't do the testing. Yet, we have 100% pass rate. My sources within the course suggest that our students consistently do better than the nurses in the course and better than the majority of physicians in the course. That's because they are told before they come into our program that they will have to take and pass this course. It's mandatory for graduation, and we prep them for it from Day 1. And these students are really good. In fact, I just wrote down that we need to do a study and follow up on the students over the past 8 years and see how many maintain certification. I think that's important also, and I think that goes well with what Bill (Kaye) was saying about keeping after people to keep these certifications up.

Bishop: First of all, you're talking about a baccalaureate program, and you're talking about people who are in school and have the time. Suppose I turn around and suddenly say to the therapist who is already working, who may have two jobs and is doing a lot at home, "You have to do a lot of preparation." I think, unlike the case with many physicians, for whom a lot of it is review, there's more new material. It takes more work than just the 2 days.

Paul Mathews: I agree with that, but, then again, I'm coming from a lot of history in respiratory care. I've been in respiratory care for 26+ years, and I remember being one of the first persons in the state I was working in to ever draw a blood gas, and being told, "You can't do that" even though the physicians were unable to obtain the

blood. Somebody said "You go ahead and try it," and I did it. The hassle that was caused by a respiratory therapist's daring to draw arterial blood when the nurses were not allowed to was unbelievable. We had the same arguments then against our drawing blood that we're hearing now about this ACLS involvement. The arguments are the same—you don't have the background; you don't have the knowledge; you don't have the practice. We learned from the results of a 1990 AARC survey of all U.S. hospitals listed by the American Hospital Association that respiratory therapists now draw the majority of blood gases in this country. We believe, further, that they will continue to do so in the future.¹ In the AARC survey, it was reported that 70-80% of all blood gases are drawn by respiratory care practitioners. Some of whom only have 9 months to 1 year of schooling. We get out there, and we let them do that, and we let them do it under a mentoring program. We give them the knowledge that they need for the specific task. You don't have to be able to name every vein and artery in the body in order to draw blood. As technology and techniques cascade from specialized, isolated use to general broad use, the need for in-depth knowledge of theory declines. How many of us can describe the design of and build a microwave oven, for example?

1. AARC Consensus Conference on Educational Direction. Year 2001: Delineating the Educational Direction for the Respiratory Care Practitioner. Proceedings, Oct 2-4, 1992, Dallas TX. American Association for Respiratory Care 1993:48.

Kaye: Beginnings always take time, and it's just like back 20 years ago when the ACLS course began, there was tremendous resistance, especially on the part of physicians and nurses. You may not want to mandate that all therapists take ACLS at your hospital. You get one to try it, "Come on,

try it out." And that RCP takes the course. It's a good experience, and that person will say, "Hey it's OK, you try it next." That's one issue. Having it in the schools is fine; we have a lot of therapists out there who aren't in school, so we have to do it in-house. Secondly, I think that in many hospitals it may be perceived as a new role. I don't think so. But if the RCPs are involved in a multidisciplinary course, with the physicians, house staff (if you have them), the nurses, they will become recognized as members of the same team. It also means that the RCPs need to be encouraged to be assertive in a positive sense. If the airway is under control, give it to somebody else and say, "Hey, can I defibrillate? I took ACLS, let me try it." I think that over time that's going to happen. Finally, even though I talked about the problems with arrhythmias and drugs, the new course is supposed to downplay a lot of that. V-fib is the easiest—it's a squiggly line. You have to make sure it's not artifact. But if the patient looks dead and it's a squiggly line, that's V-fib. With the tachyarrhythmias, it's complicated, but it's really very simple. If they're unstable, no matter what it is, you cardiovert them. If they're stable, well, try adenosine, that's OK. Try lidocaine, that's good. Just don't use verapamil if you think it's V-tach. I don't think it's that complicated. I think in the scenarios, most of us old guys are going to have to make a big change in how we teach the course. We have to be more sensitive; it's OK not to be 100% correct. If you get through and do fairly well, that's OK. If you can't get through, you don't fail. You get a—what's it called?

Fluck: "Not-yet-complete."

Paul Mathews: Not-yet-complete. And they're told not to take the complete course all over again, but to participate in those parts with which they

had problems. I think you'll get a lot of support for this from physicians and nurses. Until recently, emergency department nurses did not have much more arrhythmia or ACLS drug therapy training than did our students, and they learn!

Mickey Mathews: In the state of Kansas, they're required to hold a board certification in emergency nursing (BCEN) and be ACLS certified.

Kaye: But many of the nurses come to an ACLS course very green.

Mickey Mathews: True, true.

Kaye: And our job is to be gentle and shepherd them through.

Mickey Mathews: I agree, and there is a lot of resistance, and I've been called some nasty names because we insisted on ACLS for the emergency

department because it is the beginning of critical care when you enter the hospital. But they do come around ...

Kaye: You need to get to your Board of Medical Advisors. What they're doing is making it impossible. "No, we can't endorse ACLS for the respiratory care practitioner until we have ACLS in the schools." Well, what about those 8 zillion therapists who aren't in school anymore? They have to say "Yes, yes, yes."

Barnes: I didn't say they were reasonable about it. I just said that was their position. I do agree. I think we use the term respiratory care practitioner too loosely. If you look at other fields, to become a nurse practitioner you need a master's degree. A lot of physician assistant programs award a master's degree. I'm more of an advocate of looking at our people as respiratory therapists, and I think that's

fine. There's more than just grabbing hold of a name and saying you are a respiratory care practitioner as though that were going to open up doors for you. You have to earn that by training at a high level. ACLS certification and being a registered therapist with the NBRC—that's all well and good—but the schools have a responsibility. When they give a degree or a certificate of completion, it should imply a certain level of competence. I think the competence should extend to ACLS, regardless of whether you take an ACLS course or not. That's very important knowledge and skill area that people need to have because of what we do in hospitals. The severity of illness in the patients that we care for, the importance of identifying the prearrest condition in patients, the importance of bringing other medical staff to the bedside and starting down the road to prevent arrest from happening is really critical.

CORRECTION

On Page 273 of Marini's paper "Dyspnea during Weaning" published in the March issue of the Journal [Marini J. Dyspnea during weaning. *Respir Care* 1995;40(3):271-276], the sentence should read

"A sub-analysis indicated that for patients who have at least a moderate ventilatory drive ($P_{0.1}$ more negative than 2 cm H₂O) occlusion with a one-way valve may indeed elicit a near maximal response reliably—even without subject cooperation (Fig. 6)."

We regret the error.

Neonatal Resuscitation Program and Pediatric Advanced Life Support

Cynthia Malinowski MA RRT

- I. Introduction
- II. Neonatal Resuscitation Program
 - A. Etiology of Cardiopulmonary Arrest at Birth
 - B. Neonatal Resuscitation Program Content
 - C. Outcome of Neonatal Resuscitation
- III. Pediatric Advanced Life Support
 - A. Etiology of Cardiopulmonary Arrest in Pediatrics
 - B. Identifying a Need
 - C. Outcomes of Pediatric Resuscitation
- IV. Summary

Introduction

Educational programs that address the resuscitation of adults have been available since the early 1970s. As it became evident that newborn infants and children have unique resuscitation needs, the American Academy of Pediatrics (AAP) and the American Heart Association (AHA) suggested that guidelines be developed that were specific for these populations.¹ The call for specialized programs was supported by reports identifying a need for personnel with the necessary knowledge and skills to perform neonatal and pediatric resuscitation.^{2,3} The publication of standards for neonatal and pediatric resuscitation in 1986,⁴ served as the basis of the Neonatal Resuscitation (NRP) and the Pediatric Advanced Life Support programs (PALS). These programs were the result of a joint effort of the AHA and the AAP. The NRP and PALS courses were revised in 1992,⁵ the result of the additional years of research related to resuscitation in children.

Differences in anatomy and physiology between the pe-

diatric and adult patient have been well documented,⁶⁻⁹ and dictate an approach to resuscitation in the newborn and child that is different from that of the adult. The needs of the newly delivered patient vary from those of the older infant and child, as is acknowledged by the decision to implement two courses—one for neonatal resuscitation, NRP and one for pediatric resuscitation, PALS.

Many providers of health care for the neonate (0-28 days of age), infant (28 days-1 year of age), and child (1-8 years of age) have attended NRP and PALS. Some have initiated programs or implemented the guidelines at their facilities.¹⁰⁻¹² The results of this coordinated effort to educate respiratory care practitioners (RCPs), nurses, physicians, and paramedics in a clear, methodical fashion to optimize emergency care of neonates and children may improve the mortality and morbidity of infants and children.¹³

Although addressing the needs of the emergency- and delivery-room resuscitation environments is necessary, claiming an association between these courses and a change in survival or morbidity in newborns, infants and children, as has been demonstrated with ACLS, is difficult.

This review describes NRP and PALS, the etiology of cardiopulmonary arrest in neonates, infants and children, differences in approach for resuscitation between children and adults, current issues related to the content of the courses, and reported outcomes.

Neonatal Resuscitation Program

The NRP is designed to help clinicians prepare to care for an infant who requires resuscitation at birth—delivery resuscitation. The NRP curriculum is different from other

Ms Malinowski is Program Director, Emergency Medical Care Baccalaureate Degree Program, Department of Cardiopulmonary Sciences, School of Allied Health Professions, Loma Linda University, Loma Linda, California.

A version of this paper was presented by Ms Malinowski during the Journal Conference, "Resuscitation in Acute Care Hospitals" held in Cancun, Quintana Roo, Mexico, October 1994.

Reprints: Cynthia Malinowski MA RRT, Department of Cardiopulmonary Sciences, School of Allied Health Professions, Loma Linda University, Nichol Hall, Room 1923, Loma Linda CA 92350.

AHA life-support courses because the causes of respiratory and/or cardiac failure in infants at birth differ from those in the adult and child. The majority of delivery resuscitations are required because of premature delivery or, in the term infant, some degree of asphyxia. In most delivery resuscitations, the newborn is simply having minor difficulty in making the transition from intrauterine to extrauterine life. Because these difficulties are usually controlled by providing a neutral thermal environment and proper airway management and ventilation, airway control, preventing heat loss and providing a patent airway and ventilation are emphasized in NRP.

After delivery, most newborns respond to basic interventions, such as drying, warming, suctioning, and stimulation. Even though simple, these steps can be vital to a normal transition. The heat loss and thermogenesis that can occur after delivery are capable of initiating a sequence of physiologic events that may precipitate pulmonary hypertension and continued fetal circulation.¹⁴

The neonate who is severely asphyxiated may require an extensive resuscitation. However, mildly asphyxiated infants may need only an enriched oxygen environment for a short period of time. Some neonates with apnea typically respond well after a brief period of assisted ventilation and begin breathing on their own. And for a few, cardiac compression and drugs are required for recovery.

The educational format of NRP is also different from other AHA courses, incorporating programmed materials with lessons and posttests that are to be completed by participants prior to attending class. The class can be offered in the format of self-paced learning, group discussion, or the more traditional lesson-and-skills review over 1-2 days. The testing format requires that separate written posttests for each of the 6 individual lessons be given. Following the course introduction, short written posttests for lessons on initial steps, ventilation, intubation, chest compressions, and medications are given. Each component also uses performance checks during which the participants' skills are validated—proper warming and drying technique, administering free-flow oxygen, airway positioning, suctioning, ventilation with a bag-valve-mask system, chest compression, and intubation.

Typical medications and doses used for neonatal resuscitation in all locations (delivery room or intensive-care unit) are described, and participants are tested on medications using a written test and/or an optional Megacode practicum. Participants may repeat tests on which they were unsuccessful. Defibrillation and vessel-cannulation techniques are not included. Videotapes for review of each skills lesson are available for purchase.

Another unique aspect of the NRP is that it is not mandatory that all students be tested on all of the stations, but only on those that appear relevant to their job. For ex-

ample, a nurse may not be expected to pass the intubation-skills station and a respiratory therapist may not be asked to take the medications posttest. In this way, training time may be saved for those unlikely to be involved in a particular aspect of resuscitation but should not lead to the exclusion of any category of health-care provider from learning any of the NRP content. The best individual for a particular role during resuscitation is the person who is first available, most skilled and/or experienced, and properly educated, and may mean that a nurse needs to learn to intubate and an RCP needs to learn the proper dose of endotracheally administered epinephrine.

The AAP recommends that someone who is trained in neonatal resuscitation be present at every delivery.⁵ Although some high-risk deliveries are identified prenatally, many escape detection. A recent report suggests that only 56% of deliveries that result in the need for aggressive neonatal resuscitation are anticipated.¹⁵ Because of the uncertainty of predicting the need for a delivery room resuscitation, the AAP also recommends that qualified personnel be available in the hospital at all times. Because RCPs typically respond to all resuscitation calls in the hospital environment, they should be NRP trained, even if their hospital does not have a comprehensive neonatal intensive care unit. Considering that the obstetrician and obstetrics nurse are attending to the mother as their primary patient, it is conceivable that on occasion, the RCP will be more experienced in airway control and intubation of the newborn than any of the other personnel attending the delivery. This may be particularly true at smaller community hospitals during the night shift. The NRP may be conducted to train the RCP in the techniques, rationale, and organizational principles of newborn resuscitation.

According to the AAP and the AHA, more than 500,000 health-care providers have participated in the NRP.¹⁶ This massive education effort involved instructors from respiratory care and perinatal outreach departments and has successfully disseminated this training to practitioners over a large geographical area. In Canada, 50 health-care agencies, provincially, have participated in the program. The Canadian Council on Health Facilities Accreditation has encouraged the courses through recommendations that hospitals provide NRP training.¹⁷ The NRP has been offered throughout the world. In December, 1991 a 7-person team, with American Academy of Pediatrics representatives, trained NRP National Faculty in Romania.

Etiology of Cardiopulmonary Arrest at Birth

Only a small percentage of term newborns need extensive resuscitation. The most common diagnosis of the term newborn who requires resuscitation at delivery is asphyxia. A larger percentage of preterm than term newborns re-

quire resuscitation at birth, and these resuscitations are likely to be more extensive. Prematurity is the leading cause of mortality and morbidity in the newborn population.¹⁸ The NRP provides instruction for resuscitating both term, asphyxiated and preterm, low birthweight infants.

It is sometimes difficult to distinguish between the neonate who has suffered from prolonged asphyxia in utero, often long before delivery, and one who has a mild degree of transitional asphyxia. Infants in the former category may have low Apgar scores and bradycardia, require prolonged resuscitation at birth, and ultimately experience a poor outcome. The neonate who has been exposed to prolonged, predelivery asphyxia is suffering the effects of prenatal factors such as substance abuse that are often beyond medical control.¹⁹ Infants who experience transitional asphyxia often have no morbidity even though their initial Apgar scores were low.²⁰ Therefore, the outcome of infants with transitional asphyxia may be more influenced by resuscitation efforts than that of infants with prolonged intrauterine asphyxia.

In recent years, the etiology of perinatal asphyxia and its association with long-term outcome and medical intervention has been re-examined.²¹ Traditionally, the Apgar score has been used as a guide to identify the clinical status of the infant at delivery. It was thought to be related to the degree of perinatal asphyxia the infant had suffered, and attempts have been made to correlate it with outcomes. The Apgar score has also been used to reflect the appropriateness of medical care and to infer liability. This latter application is an unfortunate extension of the original intent because the score is unreliable as a predictor of future neurologic outcome except in extreme cases where it is persistently low, < 3 at 20 minutes.²² Studies have shown that of infants with 1-minute Apgar scores < 3 (long considered a sign of severe asphyxia), an unpredictable, but large number survive and develop normally.^{23,24} Health-care professionals whose efforts have been blamed for poor outcomes such as cerebral palsy and mental retardation in these infants, have maintained that the sequence of physiologic events leading to these tragic results may be initiated well before labor starts. Because it is difficult to distinguish between the mildly asphyxiated and severely asphyxiated newborn at birth, the neonatal resuscitation guidelines suggest a sequence of steps that are indicated for either group. The individual infant's response to these steps help to predict the degree of asphyxia suffered. The newborn who has mild transitional asphyxia also responds quickly with improvement in physiologic variables, whereas the severely asphyxiated newborn does not. These issues are relevant to the neonatal resuscitation education efforts, in that the approach is the same for any infant who has respiratory and/or cardiac depression at birth.

Recognizing that there are some exceptions to the two common reasons for depression at birth, the NRP incorpo-

rates an alternate strategy to treat infants with respiratory depression due to gestational narcotic exposure, delivery through meconium-stained amniotic fluid, and maternal blood loss. Narcan, a narcotic antagonist should be given to the infant born to a mother who has ingested narcotics within 4 hours of delivery. The infant who is delivered through meconium-stained amniotic fluid should be intubated and suctioned before ventilation is attempted, and the infant whose mother has had substantial blood loss may need a volume expander. Although the general sequence of resuscitation steps does not change, these extra steps may need to be inserted to adequately resuscitate infants, such as these.

A report on natality statistics reveals that the consequences of prematurity are important causes of death in the infant period—the effects of prematurity extend well beyond the neonatal period. The incidence of preterm birth has been steadily increasing for the last decade, and the leading causes of death of infants, other than congenital anomalies, are short gestation, low birthweight and etiologies related to perinatal asphyxia.²⁵ Although a large portion of the NRP deals with airway control and ventilation that may be applied to the term or preterm infant, the updated version of the course incorporates a special section on the resuscitation of the low birthweight newborn.

Neonatal Resuscitation Program Content

When the NRP was initiated, a logical sequence of steps based on research, authoritative anecdotal data, and, occasionally, extrapolations from adult resuscitation programs was developed so that a large-scale, cohesive education process could be implemented in a timely manner. As more research on neonatal resuscitation was reported, improvements were made to the NRP. However, the literature related to the resuscitation of the newborn remains deficient. This review cannot address all of the neonatal resuscitation issues in depth, but some specific topics deserve attention.

Initial Steps. Although this section of the course seems elementary to the sophisticated provider of neonatal intensive care, the use of the protocol contained therein may contribute to reduction in morbidity and promote a smooth transition from uterine to extrauterine life. (1) Immediately after birth, the newborn should be placed under a radiant heat source and dried and all wet linen removed. One of the benefits of administering the NRP to a diverse group of obstetric-care providers is that the potentially disastrous consequences of cold stress in the newborn is shared with the caregivers who have the best opportunity to prevent morbidity from using these simple steps. (2) The newborn should be placed in a supine position with the airway opened, and the nares and mouth should be suctioned with

a bulb syringe. The majority of apneic neonates will be stimulated by these activities and initiate spontaneous respirations. However, if they do not, tactile stimulation of the heels or back may stimulate the infant to breathe. The attempt to initiate breathing by tactile stimulation should be brief because if the newborn is going to respond, he will do so within a few seconds. If apnea persists, ventilation with a bag-valve-mask is required.

Ventilation and Compressions. It has become increasingly clear that for the neonate who has difficulty with transition to extrauterine life, adequate ventilation is an immediate priority. The bradycardia sometimes associated with cardiopulmonary distress at birth often improves quickly with ventilation. For caregivers, some practice and supervised experience may be required to adequately ventilate the newborn with a bag-valve-mask. Ventilation difficulties include nasopharyngeal secretions, the presence of fluid on the face preventing an adequate mask seal, and the inability to estimate the proper ventilating pressure necessary for lung expansion.

Although the use of simultaneous ventilation and chest compression was never clearly advocated, previous NRP guidelines contained no strong statements against it.²⁶ As in many resuscitation courses, participants were taught to concentrate on achieving perfect ratios of breaths to compression and rates of compressions and ventilation, instead of providing adequate tidal volumes—a focus that may persist in clinical setting. Data reported in adult humans and newborn animals showed no advantage to synchronized ventilation and compression.^{27,28} Various issues were raised, such as whether the pressure manometer used during bag-valve-mask ventilation reflected cardiac compressions or esophageal-sphincter-opening pressure, instead of the pressure developed in the lung.²⁹ The decision was made to emphasize the necessity of adequate ventilation even at the expense of the previously suggested optimal rate of chest compression.³⁰ Although the rate of compression for newborns remains at 120/min, the pause to allow delivery of an adequate breath reduces the compression rate to 90/min and maintains the ventilation rate at 30/min. No data suggest that the reduced compression rate is detrimental to the infant, and the emphasis on appropriate tidal volumes during resuscitation may offer some benefit. When chest compressions are not required, the ventilation rate should be 40-60 breaths/min not 40 breaths/min as given in the previous guidelines.

Medications. The information provided in the NRP on medication-use during neonatal resuscitation is far less than that provided in ACLS and PALS. Only epinephrine, sodium bicarbonate, and naloxone hydrochloride are covered in the NRP. Volume expanders are covered in a separate sec-

tion. Other drugs are rarely required for delivery-room resuscitations, and those used to manage infants in the neonatal intensive care unit are not addressed in the NRP.

Extrapolation of medication doses from adult-human and animal studies to neonates may not be appropriate because the physiology of newborns is unique. Discrepancies between adult and newborn animal models exist regarding the actions and appropriate doses of drugs.³¹ A fluid-filled or partially fluid-filled lung may enhance the absorption of endotracheally delivered drugs.³² Liver and kidney function may affect the ability of the newborn to process medications. The absorption, metabolism, and excretion of drugs are not the same in the newborn as in the adult. This is especially true in premature newborns and those who are critically ill.³³ In addition, the complications from the effects of certain drugs can be different in newborns. A profound complication may result from the newborn's immature brain structure, particularly in the highly vascular germinal matrix, an area prone to hemorrhage.³⁴ Changes in blood flow to the brain or blood osmolality is especially dangerous. Because cerebral-blood-flow regulation is immature in premature neonates, they are especially sensitive to brain-blood-flow fluctuations and blood pressure changes that may result from the use of resuscitation drugs.

Data conflict regarding the use of medications during newborn resuscitations. The results of research and the recommendations related to the use of high-dose epinephrine (HDE) in the adult literature exemplify such conflicts. In adult studies, although there are controversies in the use of HDE, the ACLS guidelines allow individuals to make independent decisions on its use and, once the initial dose is given, administer the higher doses of epinephrine.³⁵ The terminal cardiac activity found in neonates is different from that found in adults. Although the adult who requires resuscitation is often discovered having ventricular fibrillation, 80% of neonates who need resuscitation have bradycardia.³⁶ The use of HDE in the literature is often in a model with ventricular fibrillation. In theory, epinephrine should be effective for the resuscitation of bradycardia and asystole, and a limited report of pediatric cases showed some success with this strategy.³⁷ More evidence from newborn animal models and clinical trials is needed before the questions regarding HDE are answered and before HDE can safely be recommended for newborn resuscitation. Currently (1995), the NRP textbook does not recommend any change in epinephrine dose.³⁸

Endotracheal-epinephrine use in neonatal resuscitation is also perplexing. Recommendations for the epinephrine dose administered by the endotracheal route evolved based on the adult literature. That the endotracheal dose is larger than the intravenous dose is based on a study that demonstrated inadequate epinephrine absorption after endotracheal administration.³⁹ However, Lucas et al⁴⁰ found that in the newborn

lamb the hypoxia-induced low pulmonary blood flow usually associated with the low-cardiac-output state encountered in newborn resuscitation did not lead to impaired absorption of intratracheal epinephrine (Fig. 1). The most recently published PALS textbook⁵ and the 1992 NRP update¹⁶ suggest that giving epinephrine by the endotracheal route may lead to low and potentially inadequate serum concentrations of epinephrine. Lucas and colleagues⁴⁰ urge caution when administering higher than previously established doses for endotracheal epinephrine in the newborn.

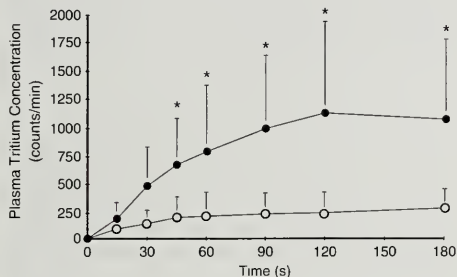


Fig. 1. Mean (+ SD) arterial plasma tritium (counts/min) in 6 lambs following endotracheal administration of epinephrine during low pulmonary blood flow (●) and normal pulmonary blood flow (○). Overall, significantly higher tritium counts were obtained during low pulmonary flow than during normal pulmonary flow ($p = 0.02$). * $p < 0.03$ for comparisons at individual time periods. (From Reference 40, with permission.)

As is true in ACLS and PALS, the use of sodium bicarbonate in newborn resuscitation has largely fallen out of favor. The justification for decreased use of sodium bicarbonate in pediatric and adult resuscitation holds for the newborn as well, and some additional concerns are specific to neonates. The theory that bicarbonate administration transiently leads to increases in intracellular CO_2 levels and pH reduction is equally cogent in the newborn and in the adult. In addition, the use of hyperosmolar and hypertonic solutions such as sodium bicarbonate can lead to fluid shifts, which can ultimately result in cerebral hemorrhage and edema (Table 1).⁴¹

Table 1. Problems with Sodium Bicarbonate Therapy in Neonates

Problem	Consequence
Hyperosmolality	Brain plasma H_2O shifts, cerebral edema
Hypertonicity	Intraventricular hemorrhage
Low perfusion	Intracellular acidosis, myocardial ineffectiveness

Meconium-Stained Amniotic Fluid. The treatment of the newborn who is delivered through meconium-stained amniotic fluid is another controversial issue. The presence of

meconium (fetal fecal material) in amniotic fluid has long been associated with in-utero asphyxia. The presence of meconium in the lung, inhaled during or after delivery, has been thought to be responsible for the severe lung parenchymal changes that may lead to respiratory distress and even failure. In 1974, Gregory et al⁴² concluded that immediate tracheal intubation followed by suctioning, using the endotracheal tube as a suction catheter, was necessary to prevent meconium, found in the nose and oropharynx at birth, from being inhaled into the lung periphery. The combined treatment of upper-airway suctioning as the infant's head is delivered and immediate intubation and suction after delivery decreased mortality and morbidity in their patients. The initial standards for the neonatal resuscitation program called for immediate intubation and suctioning of those infants in whom the amniotic fluid was stained with thick meconium. Thin or yellowish meconium may not be a threat to lung function.

More recently, the studies that were initially done in the 1970s using tracheal suction were duplicated with some minor, but important, changes. Linder et al⁴³ examined 572 infants delivered through meconium (Fig. 2). Infants who had Apgar scores ≤ 8 were excluded thereby excluding all patients who showed no initial signs of severe asphyxia. Two groups were studied—infants who all were immediately intubated and those who were managed without intubation. The infants receiving the traditional care of intubation and suction ($n = 308$) had a higher percentage of respiratory complications than those who were not intubated.

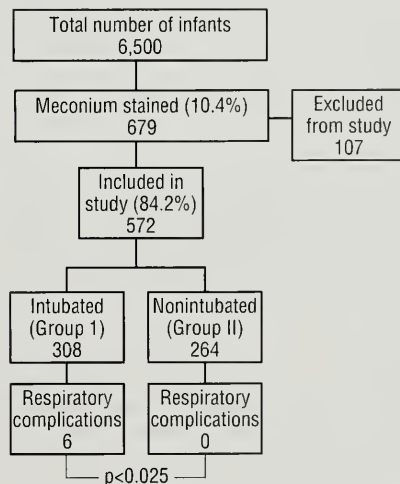


Fig. 2 Distribution of mortality and morbidity among intubated and nonintubated infants delivered through meconium-stained amniotic fluid. (From Reference 43, with permission.)

These complications included meconium aspiration syndrome ($n = 4$) and laryngeal stridor ($n = 2$). In the nonintubated group ($n = 264$) no infant suffered any respiratory complications. In the entire series there were no deaths—not surprising because these babies were all rather vigorous. Although this study was criticized for some methodologic flaws (including the use of endotracheal tubes that were too small) it did raise some interesting questions. Even in the presence of thick meconium and no suction, why did all babies survive with no respiratory sequelae? Why do some newborns develop meconium-aspiration syndrome and die despite prompt intervention and aggressive tracheal suctioning? Why do those who are delivered through thick, pea-soup meconium with little suction before their first breath have no apparent respiratory distress?

A study in animals⁴⁴ has verified that meconium is inhaled deep into the lung periphery prior to delivery in the fetus who is severely asphyxiated. These data tend to confirm the conclusion of Linder and colleagues⁴³ that postdelivery suctioning may not offer as great an advantage as previously thought. In animals, asphyxia without the presence of meconium led to pathologic and radiologic findings similar to those believed to be caused by meconium aspiration alone. Meconium in the unasphyxiated animal lung caused only minor changes that were easily overcome by the healthy newborn.⁴⁵ Thus, the severe pathology that causes respiratory distress and failure in the newborn delivered through meconium-stained amniotic fluid may be more related to the lung damage due to in-utero asphyxia and less related to the effects of inhaled meconium. In the babies studied by Linder's group,⁴³ there may have been little or no lung damage due to prenatal asphyxia. These "healthy" babies may have had no difficulty in clearing meconium from their peripheral airways.

This controversy cannot be easily solved, and it would be premature to change the practice of suctioning neonates born through thick meconium-stained amniotic fluid; especially those who are not vigorous and who have low Apgar scores might benefit from suctioning to remove meconium from their airways. The new NRP guidelines allow avoidance of tracheal suction in the vigorous unasphyxiated neonate, giving the benefit of the doubt to this evolving practice.

Outcome of Neonatal Resuscitation

Although the presence of an organized perinatal services delivery system and staff specially trained in neonatal care can improve mortality during transport,⁴⁶ the effect of the NRP implementation on mortality or morbidity is not known. Documentation of errors during the delivery resuscitation of the newborn confirms the value of this program.⁴⁷ However, good research is lacking and its conduct problematic. To carry out a randomized, prospective study and with-

hold NRP from potential providers of delivery resuscitation may not be ethical. On the other hand, research that uses controls from a time period when NRP was not available may not be valid because of advances in neonatal care (eg, exogenous surfactant therapy) that may confound the results and prevent strong conclusions from being drawn.

A small report, written as a letter, from a physician who attempted to reduce infant mortality in a poor rural province in Yinshan County, People's Republic of China illustrates the potential impact of NRP on infant mortality.⁴⁸ Even though neonatal intensive care is unavailable, the training of maternal and child-health personnel in the concepts of the NRP reduced the death rate from asphyxia from 7.1% to 0.45% in 6 months.

Pediatric Advanced Life Support

The PALS course is similar to the ACLS course in scope, curriculum, and traditional course schedule. However, PALS's approach is different because the etiologies for cardiopulmonary arrest and the anatomy and physiology in the infant and child, are unique. In the U.S., more than 50,000 providers have taken PALS, and it has been offered in other countries.¹⁶

Etiology of Cardiopulmonary Arrest in Pediatrics

Whereas most of the cases of cardiac arrest in adults are due to cardiac pathophysiology, in children most resuscitations are required because of severe hypoxemia and acidemia that can occur from both respiratory and cardiac causes. The majority of resuscitations in pediatrics occur in infants younger than 1 year of age.⁴⁹ Apparent life-threatening events such as sudden infant death syndrome, sepsis, airway obstruction, congenital anomalies, complications related to birth, and intentional injury are commonly encountered reasons for infants to require resuscitation.^{25,50} Injury, intentional and unintentional, is the greatest cause of death in children over the age of 1 year (Table 2). It has been estimated that 20,000 children will have died in 1994 as a result of injury.⁵¹ In the pediatric patient, trauma accounts for most childhood-acquired disability.⁵²

Table 2. Common Causes of Pediatric Injury

Blunt Trauma	Child abuse
Motor vehicle accident	Penetrating injuries
Bicycle mishap	Burns and smoke inhalation
Pedestrian related	Toxic substances
Falls	Drowning

Children who arrive at the emergency department apneic and pulseless rarely survive neurologically intact.^{53,54} Although the statistics vary as to the outcome of adults in

similar states of cardiorespiratory arrest, it appears that adults fare better than children.⁵⁵⁻⁵⁷ This can be partially explained by the nature of the "secondary cardiac arrest" commonly seen in children compared to that commonly seen in adults. In the adult, cardiac arrest is most often caused by a primary cardiovascular problem, such as coronary artery disease, that results in a myocardial infarction. Prompt bystander CPR and rapid ACLS preserve the function of other organs. However, the child rarely has an initial cardiac episode leading to arrest but, rather, develops a sequence of prolonged hypoxemia, acidemia, and ischemia that affects many organs prior to an actual cardiac arrest. By the time a child's heart fails, he has sustained irreversible multiple organ damage. In children, asystole is the cardiac rhythm that usually follows this sequence of hypoxemia and acidemia.¹ Ventricular fibrillation is the most common arrest rhythm in the adult. The outcome of the resuscitation of a child in asystole is usually dismal. However, prompt resuscitation of children with asystole may still be associated with a better outcome than asystole in the adult.⁵⁸

The most advantageous approach to the critically ill infant and child is to recognize impending respiratory and cardiac decompensation at an early stage, prior to the development of severe levels of acidosis that can lead to cardiorespiratory arrest. The educational process that may result in the most improvement in outcome is training those in the chain of medical care—including paramedics, pediatricians, and emergency-department staff—to recognize early signs of respiratory distress and hypotension. As in the neonate, the prognosis for the child with apnea *not* associated with cardiac arrest is good and the treatment of impending respiratory failure has the best prognosis of all.^{53,55}

In a review of the etiology and management of critical illness and injury in a pediatric emergency department, the survival of children who were apneic and pulseless was 10.3%, compared to patients with apnea only of whom 96.9% survived.⁴⁹ This report reinforces the importance of teaching appropriate airway management for the emergency treatment of children.

Identifying a Need

Seidel et al.^{3,59} from the Ad Hoc Committee on Care of the Critically Ill Child for a Los Angeles chapter of the American Academy of Pediatrics published a 2-part report on emergency care for children in 1984. Emergency medical services for children in Los Angeles County were reviewed. During the establishment of previously published guidelines in 1980 for prehospital management of pediatric patients, the committee discovered that little trauma research related to emergency care of children had been published. Seidel's group found that children who suffered from trauma had a higher mortality rate than adults.

Prehospital providers were ill-equipped and uneducated in the emergency treatment of the pediatric patient. The death rate of children involved in a traumatic accident were higher in areas where there were no specialized pediatric centers. Surveys were sent to 98 randomly selected EMT/paramedic training programs in the United States. Respondents were asked about their curriculum for pediatric patients. This survey showed that most programs for paramedics have fewer than 10 hours out of 400-2,000 hours of didactic or clinical training in pediatrics even though the pediatric patient population accounted for 10% of their workload. The deficits in education were identified (Table 3⁵⁹). In addition, the appropriate equipment and supplies for resuscitation of infants and children were lacking on emergency vehicles. Another group identified the need for pediatric resuscitation education using the results of a survey of pediatricians.⁶⁰ This study demonstrated their lack of preparedness for pediatric resuscitation.

Table 3. Content Deficiencies of Training Programs Surveyed in Pediatric Prehospital Care*

Topic	Programs without Topic in Curriculum (%)
Pediatric field simulations	50
Pediatric dysrhythmias	50
Envenomation	45
Pediatric hypotension	36
Approach to the comatose child	29
Drowning	26
Sexual abuse	24
Advanced pediatric life support	22
Diarrhea and dehydration	22
Neonatal resuscitation	16
Pediatric trauma	14

* From Reference 59, with permission

The challenge was clear—to construct a course that addressed the care of the critically ill child by teaching practitioners to (1) identify early signs of impending respiratory and cardiac failure from a wide range of etiologies and (2) care for the pediatric trauma patient.

Description of course. The Pediatric Advanced Life Support course is conducted with lectures on pharmacology, rhythm disturbances and their management, and recognition of respiratory failure, cardiopulmonary arrest, and shock. A section on newborn resuscitation focuses on the emergency department delivery and resuscitation. These guidelines for newborn resuscitation are similar to those found in the NRP although less instruction is devoted to individual topics such as intubation. Skill stations similar to the ACLS stations are used but with child-sized man-

nequins. Lessons in vascular-access techniques allow for lectures and practice in intraosseous puncture and vascular infusions. Testing is accomplished both by a comprehensive written examination and skills stations. A suggested but not mandatory topic, critical stress debriefing, may have an important place in helping the health-care provider cope with tragic outcomes of resuscitations. In the most recently published PALS textbook,¹⁶ a section has been added that addresses the special needs of the pediatric trauma patient who requires resuscitation.

Content related to trauma in the child has recently been added to the new guidelines.¹⁶ This section describes the pediatric trauma score (a modification of the Glasgow Coma Scale) for infants and children, emphasizes cervical spine immobilization, and provides details about hypovolemic shock. A recent report confirms that penetrating injuries such as gunshot wounds are increasingly common in children, including young toddlers.⁵¹ The unique characteristics of injuries like these are likely to be addressed in more detail in the future.

Another important topic, avoidable accidental injury in the child, must not be ignored. An accident prevention program, the Pediatric Basic Life Support Program, is available nationally and should be made accessible to parents and caretakers of children.

Anatomy and Physiology in Children. The anatomy and physiology of the child are different from that of the adult and, therefore, patterns of injury, assessment techniques, and treatment are different from that of the adult. Although a comprehensive discussion is not appropriate, a few of these differences are worth mentioning with regard to resuscitation.

The child's head is the most common site of pediatric injury. Multiple system injuries are more likely to occur in the child than in the adult because of the ratio of the traumatic forces to the child's smaller body. These injuries can be unsuspected because a lack of evidence of trauma on the child's external body surface is common. The result of injury to a child's internal organs can be greater than in the adult because the child has a smaller mass of protective anatomy, such as muscle and subcutaneous tissue.⁶¹

The components of a correct neurologic assessment vary depending on the age of the patient, and the clinician needs to know the common behavioral and neurologic signs of the young infant and the older child. The young infant's neurologic function is assessed by observing eye contact with parents and eye movement following toys or wiggling fingers across the field of vision. The untrained evaluator may have trouble discerning the level of alertness in young children. A subtle change in mentation in an 8-month-old child could be a serious sign of injury or hypoxemia. The role of the parents as evaluators is important in the young child. A parent's

assessment as to whether the child is "acting as himself" can be an important part of the medical evaluation.

The clinician must be alert for the signs of abdominal injury after a traumatic accident. However, when children are frightened they may swallow large amounts of air, resulting in confusing abdominal symptoms and increased risk of aspiration.

Hypothermia and dehydration due to the infant's increased surface area-to-body weight ratio may exacerbate his cardiopulmonary demise. The pediatric patient's kidneys are less able to change the dilution and concentration of urine to prevent water loss. Differences in the anatomy of the child's airway are important when making decisions as to the urgency and performance of endotracheal intubation.

Course Content. Tachycardia and tachypnea are hallmarks of impending cardiopulmonary failure in the child. Hypotension may not be present in the pediatric patient until irreversible physiologic events have already taken place, its presence indicates a critical emergency. The child is able to maintain his normal blood pressure for a period of time; this phase is called, compensated shock.⁶² Recognizing this first phase of shock may enable earlier intervention and prevent cardiovascular collapse, and recognizing signs and symptoms of impending hypotension (slow capillary refill, cold skin temperature, low urine output, and alterations in mental state) may be more frequently life-saving than in an adult. In cases of severe hemorrhage, large blood loss can occur before hypotension can be documented. A pediatric trauma patient's blood volume may decrease by 30-40% before his blood pressure falls below normal.⁶³

Respiratory assessment and intervention are an important emphasis for any educational program in emergency care of the child. The recognition of signs of respiratory failure such as tachypnea, absent or diminished breath sounds, retractions, nasal flaring, head bobbing and level of consciousness are especially emphasized because it is usually difficult to obtain arterial blood-gas values in children to quantify respiratory failure. With proper airway management, apnea not associated with cardiac arrest can have an excellent outcome.^{53,55}

Because pediatric resuscitations encompass a variety of age groups, the provider of pediatric emergency care must be familiar with a variety of normal ranges for vital signs and behavior.

Vascular access is important in the child who is suffering from impending cardiopulmonary collapse or receiving resuscitation. The pediatric patient's peripheral veins are difficult to cannulate, and prolonged attempts to do so may delay resuscitation unnecessarily. In the pediatric trauma center or the intensive care unit, staff who are highly experienced in establishing an intravenous line (I.V.) may have less difficulty. Rosetti et al⁶⁴ reported that, in children, expe-

rienced emergency department personnel could require in excess of 10 minutes to start an I.V. and were never able to obtain vascular access in 6% of them. Partly through the education received in the PALS course, many providers of pediatric emergency care are being trained in intraosseous cannulation, a method that employs infusion into the intramedullary sinus of the tibia (Fig. 3). Schoenfeld et al⁴⁹ studied emergency interventions with pediatric patients and found that 24% of children had vascular access established through tibial intraosseous lines. This alternative method of vascular access is safe, reliable, takes a short time to accomplish, and is successful in the prehospital and in-hospital resuscitation.⁶⁵ Intraosseous cannulation has become fairly routine in pediatric emergency care in some locations.

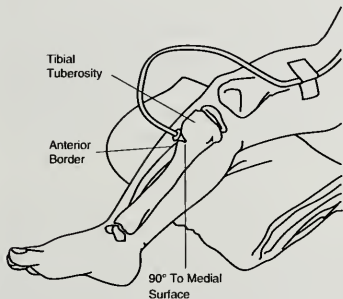


Fig. 3. Technique for drug administration by the intraosseous route during pediatric resuscitation. (From Reference 16, with permission.)

The fluid therapy and medication section of the PALS textbook is similar to ACLS and more comprehensive than is that of the NRP. There is a review of cardiac rhythm disturbances and information related to defibrillation and cardioversion, although it is rare that children require either.

Outcomes of Pediatric Resuscitation

The documentation that educational components of PALS and NRP result in changes in outcome for neonates, infants, and children is lacking. The rationale that developed supporting the implementation of these programs was sound, but studies demonstrating that these programs have had a clear impact on resuscitation practices and emergency care for children have not been published. Questions about the cost of institution of these curricula have been raised and interpretations of the data regarding specialized pediatric training conflict. Although the need for improvement in resuscitation in children is clear, those who provide prehospital care to children advocate these programs even though the rationale for their use has been based largely on theory.

One controversy regarding specialty training of

providers in pediatric resuscitation is reflected in the studies of prehospital pediatric emergency care. There appears to be a clear rationale for optimization of oxygen transport in the child before cardiopulmonary collapse has occurred. In a review of the cases of 2 children who required out-of-hospital resuscitation, Chameides⁶⁶ concludes that prehospital care may affect "meaningful" outcomes in infants and children who require CPR.

Many have called for early and aggressive intervention and endotracheal intubation in children with prehospital cardiopulmonary arrest. There have been conflicting reports about intubation performed in children, prior to intensive-care unit admission, and the conflict may relate to the amount of training the emergency and prehospital staff had received in pediatric airway management. The reduced percentage of successful intubations in children as compared to adults is a concern. Aijian, et al⁶⁷ conducted a retrospective study of all prehospital victims of medical cardiopulmonary arrest under the age of 19 years. The success rate for paramedics was 64%, considerably less than the 86% to 97% rate of success of this group in adults.^{68,69} The report also stated that intubation was attempted in children only 66% of the time. This result was attributed to either the lack of confidence of paramedics who had little previous experience or to their success with bag-valve-mask ventilation. These factors were not ranked in order of importance. The paramedics in this system had not received any specialized pediatric training, such as PALS, which may have made them less confident in both making a decision to intubate and attempting the procedure. The difference in survival to hospital admission between nonintubated and intubated children was not statistically significant and was generally dismal (2 out of 63 patients survived).

In another example of an attempt to quantitate paramedics' ability to successfully intubate pediatric patients, Losek, et al.⁵⁰ reviewed the success rate of Milwaukee County paramedics. They reported an overall 78% success rate that increased to 93% in pulseless non-breathing patients. Some of the complications and intubation difficulties that were reported could be remedied with proper training. For example, several infants arrived in the emergency room with a size 2.5 mm ID endotracheal tube. Such tubes are too small, illustrating the providers' lack of familiarity with appropriately sized endotracheal tubes. Problems with intubation included the inability to visualize the glottis and vocal cords because of mucus and vomitus. This difficulty may be remedied by emphasizing the availability and use of suction equipment.

In another review of emergency intubation, Nakayama et al⁷⁰ focused on injured infants and children. Infants and children intubated in the prehospital and emergency department setting were reviewed. The intubators were paramedics and physicians. A high complication rate relat-

ed to emergency intubation was reported. Intubation attempts were described as multiple, unsuccessful, and associated with airway complications (Table 4). This result is a concern. However, further examination of the patient population that was studied and the circumstances under which intubation was attempted may explain the seemingly poor outcome. Ninety percent of the patients had head injuries and 75% were comatose. Current recommendations for early endotracheal intubation and assisted ventilation in the head-injured child were outlined in that study, supporting the decision on the part of the paramedic and physician to intubate. Some complications may have been due to the out-of-hospital conditions under which the intubations took place. These patients all suffered from trauma, the most complicated and challenging out-of-hospital environment in which to treat the critically injured child.

Table 4. Distribution of Airway Complications by Treatment Site in Pediatric Trauma Patients*

Complication	Number	Site†	Age (years)	Deaths
Right main stem intubation	5	1S, 3R, 1C	1, 4, 4, 7, 8	0
Massive subcutaneous emphysema from cricothyroidotomy needle	2	2S	12, 15	2
Esophageal intubation	1	S	1.5 months	1
Failed nasotracheal intubation in an open skull fracture	1	S	4.5	1
Massive aspiration	1	R	15	0
Bilateral vocal cord paralysis	1	R	6	0
Subglottic stenosis	1	R	4	0
Failure of adequate preoxygenation	2	2C	4, 10	0
Extubation during transport	1	S	8	0
Left vocal cord paralysis	1	C	5 months	0
Total	16	6S, 6R, 4C	mean 6.45	4

* From Reference 58, with permission.

† Location of intubation: S, scene of injury; R, referring hospital; C, Children's Hospital.

Prehospital care of the critically injured infant or child has environmental constraints not found in the emergency room or intensive-care unit. Inability to properly position the patient, inadequate light, the need to control crowds, limited equipment for multiple accident victims, interference by hysterical family members, too few trained personnel, and the lack of use of paralytic and sedating agents may contribute to intubation failures. An esophageal or right mainstem intubation may not be identified in the child

whose chest remains out of view, trapped in a vehicle. The children in this series had serious multisystem injuries, which could have contributed to the stress and urgency under which the paramedics and physicians performed. The author of this study emphasized the need for education and training for all providers (physician, paramedic, nurse, and RCP) of care to acutely injured children.⁷⁰

In another examination of outcomes of pediatric resuscitation, Quan et al,⁵⁸ had a comparatively high success rate in resuscitation of apneic, pulseless victims of near drowning. The 21% average success rate was comparable with adult cardiac arrest outcomes following prehospital ACLS. They suggested that this finding may be partly attributed to the submersion victims being in cardiopulmonary arrest prior to severe multisystem failure. The emergency services in Seattle, where this study was conducted, have an efficient and aggressive prehospital system, and a larger percentage of children are intubated in the field, unlike studies from cities reporting poorer survival. The authors stated that their findings support the need for aggressive prehospital care of children.

Summary

The need for delivery resuscitation of the newborn cannot be predicted in most cases; therefore it is judicious to train all providers who may be involved in the delivery of newborns to follow guidelines developed to improve outcome, especially in the presence of transitional asphyxia. The Neonatal Resuscitation Program emphasizes basic steps of warming, drying, suctioning, and adequately ventilating the newborn. It also addresses current theories regarding resuscitation of the low birthweight newborns, infants with meconium aspiration, and medication use. The NRP applies to all acute-care hospitals that provide delivery services and those at which a respiratory therapist is likely to be present in the high-risk delivery or unanticipated delivery-room resuscitation. Outcomes have not been well documented and more clinical research is needed to identify which therapeutic strategies promote the best survival in this population.

A topic that should be included in the NRP of the future is exogenous surfactant delivery. Respiratory distress syndrome has been a significant cause of death and morbidity in prematurely born neonates. Exogenous surfactant therapy has had a dramatic effect on the death rate of premature infants and on the incidence of respiratory distress syndrome. Current methods of surfactant administration demand that personnel proficient in management of the low birthweight newborn be present. As hospitals with all levels of nurseries continue to receive the prematurely delivered newborn and better methods to administer surfactant are discovered, the NRP could add information and a skills laboratory on surfactant administration.

A trained cadre of health professionals who are proficient in the specific resuscitation skills required in pediatric patients can make a difference. The infant and child have different anatomy, physiology, and disease etiology that need to be emphasized and understood by the pediatric caregiver. The Pediatric Advanced Life Support course focuses on early recognition and treatment of respiratory failure and shock. The possibility of improving outcome with properly trained prehospital providers is a topic of interest. Intubation in the field can be successful and may contribute to a reduction in mortality, although complications of field intubation need to be reduced.

REFERENCES

- Eisenberg M, Bergner L, Hallstrom A. Epidemiology of cardiac arrest and resuscitation in children. *Ann Emerg Med* 1983;12:672-674.
- Myers S, Fisher D, Moawad A, Paton J, Lee Kwang-Sun, Ferguson M. Assessment of potentially avoidable perinatal mortality in a regionalized program. *J Reprod Med* 1990;35:29-34.
- Seidel J, Hornbein M, Yoshiyama K, Kuznets D, Finklestein J, St Geme J. Emergency Medical Services and the Pediatric Patient: Are the needs being met? *Pediatrics* 1984;73:769-772.
- Emergency Cardiac Care Committee and Subcommittees, American Heart Association. Neonatal Advanced Life Support. *JAMA* 1986;225:2969-2973.
- Emergency Cardiac Care Committee and Subcommittees, American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiac care, part V-VII. *JAMA* 1992;268(16):2171-2302.
- Karlberg P. The adaptive changes in the immediate postnatal period, with particular reference to respiration. *J Pediatr* 1960;56:585-604.
- Milner A, Vyas H. Lung expansion at birth. *J Pediatr* 1982;101:879-876.
- Adamson K, Gandy G, James L. The influence of thermal factors upon oxygen consumption of the newborn human infant. *J Pediatr* 1965;66:495-508.
- Berry F, Yemen T. Pediatric airway in health and disease. *Pediatr Clin North Am* 1994;41:153-180.
- Singhal N, McMillian D, Lockyer J, Gondocz S, Shukla A, Swaby C. Attitudinal and resource changes after a neonatal resuscitation training program. *Neonatal Network* 1992;11:37-40.
- McCulloch K, Vidyasagar D. Assessing adherence to standards for neonatal resuscitation taught throughout the perinatal referral area. A quality assurance activity. *Pediatr-Clin-North Am* 1993;40:431-438.
- Taylor M, Soud T. Florida Pediatric Emergency Nursing Education project. *J Emerg Nurs* 1991;17:152-155.
- Foltin G, Salomon M, Tunik M, Wecheiderman W, Treiber M. Developing prehospital advanced life support for children: The New York City experience. *Pediatr Emerg Care* 1990;6:141-144.
- Klaus M, Fanaroff A. Care of the high risk neonate. 3rd ed. Philadelphia: W.B. Saunders, 1986:100-101.
- Jain L, Vidyasagar D. Cardiopulmonary Resuscitation of Newborns. *Pediatr Clin North Am* 1993;40:287-301.
- American Heart Association, American Academy of Pediatrics. Textbook of Neonatal Advanced Life Support. Dallas and Park Ridge: 1994.
- Higgins A. Managing the distressed newborn. *Nursing BC* 1992;24:13.
- Centers for Disease Control and Prevention, U.S. Department of Health and Human Services. Advance Report of Final Natality Statistics, 1991. Monthly Vital Statistics Report. Sept. 9, 1993;42(3,Suppl):1-48.
- Bejar R, Wozniak P, Allard M, Benirschke K, Vaucher Y, Coen R, et al. Antenatal origin of neurologic damage in newborn infants. *Am J Obstet Gynecol* 1988;159:357-363.
- Williams C, Mallard C, Tan W, Gluckman P. Pathophysiology of perinatal asphyxia. *Clin. Perinatology* 1993;20:305-325.
- Marlow N. Do we need an Apgar score? (editorial). *Arch. Dis. Child.* 1992;57:785-787.
- Committee of the Fetus and Newborn. Use and abuse of the Apgar score. *Pediatrics* 1986;78:1148-1149.
- Palme-Kilander C. Method of resuscitation in low-APGAR-score newborn infants a national survey. *Acta-Paediatr* 1992;81:739-744.
- Catlin E, Carpenter M, Brann B, Mayfield S, Shaul P, Goldstein M, Oh W. The Apgar score revisited: Influence of gestational age. *J Pediatr* 1986;109:865-868.
- Center for Disease Control and Prevention - U.S. Department of Health and Human Services. Advance Report of Final Mortality Statistics, 1991. Monthly Vital Statistics Report Aug 31, 1993;42(2,Suppl):1-45.
- American Heart Association, American Academy of Pediatrics. Textbook of Neonatal Resuscitation. Dallas and Elk Grove Village:1987.
- Swenson R, Weaver W, Niskanen R, Martin J, Dahlberg S. Hemodynamics in human during conventional and experimental methods of cardiopulmonary resuscitation. *Circulation* 1988;78:630-639.
- Berkowitz J, Chantarojanasiri T, Koehler R, Schelen C, Dean J, Michael J, Rogers M, Traystman R. Blood flow during cardiopulmonary resuscitation with simultaneous compression and ventilation in infant pigs. *Pediatr Res* 1989;26:558-564.
- Melker R, Banner M. Ventilation during CPR: two-rescuer standards reappraised. *Ann Emerg Med* 1985;14:397-402.
- Burchfield D, Erenberg A, Mullett MD, Keenan WJ, Denson SE, Kattwinkel J, Bloom R. Why change the compression and ventilation rates during CPR in neonates? (letter). *Pediatrics* 1994;93(6, pp 1):1026-1027.
- Berkowitz ID, Gervais H, Schleien CL, Kochler RC, Dean JM, Traystman RJ. Epinephrine dosage effects on cerebral and myocardial blood flow in an infant swine model of cardiopulmonary resuscitation. *Anaesthesiology* 1991;75:1041-1050.
- Burchfield D, Berkowitz ID, Berg RA, Goldberg RN. Medications in neonatal resuscitation. *Ann Emerg Med* 1993; 22 (pt 2): 435-439.
- Fanaroff A, Martin R, editors. Neonatal-Perinatal medicine: diseases of the fetus and infant 5th ed. St. Louis: Mosby Yearbook, 1992 1019-1038.
- Lou HC. On the pathogenesis of germinal layer hemorrhage in the neonate. *APMIS* 1993;101 (40, Suppl): 97-102.
- Cummins R. Advanced Cardiac Life Support Textbook. American Heart Association. Dallas:1994: 7-4.
- Walsh CK, Krongrad E. Terminal cardiac electrical activity in pediatric patients. *Am J Cardiol* 1983;51:557-561.
- Goetting MG, Paradis NA. High-dose epinephrine improves outcome from pediatric cardiac arrest. *Ann Emerg Med* 1991;20: 22-26.
- American Heart Association, American Academy of Pediatrics. Textbook of neonatal resuscitation. Dallas and Elk Grove Village: 1994.
- Quinton DN, O'Byrne G, Aitkenhead AR. Comparison of endo-

- tracheal and peripheral intravenous adrenaline in cardiac arrest: is the endotracheal route reliable? *Lancet* 1987;1:828-829.
40. Lucas VW, Preziosi MP, Burchfield DJ. Epinephrine absorption following endotracheal administration: effects of hypoxia-induced low pulmonary blood flow. *Resuscitation* 1993;27: 31-34.
 41. Wheeler A, Sadri S, Gutsche B, DeVore J, David-Mian Z, Latshevsky H. Intracranial hemorrhage following intravenous administration of sodium bicarbonate or saline solution in the newborn lamb asphyxiated in utero. *Anesthesiology* 1979;51:517-521.
 42. Gregory GA, Gooding CA, Phis RH, Tooley WH. Meconium aspiration in infants: a prospective study. *J Pediatr* 1974;85:848-852.
 43. Linder N, Aranda JV, Tsur M, Matoth I, Yatsiv I, Mandelberg H, et al. Need for endotracheal intubation and suction in meconium-stained neonates. *J Pediatr* 1988;112:613-615.
 44. Duenhoelter JH, Pritchard JA. Fetal respiration: quantitative measurements of amniotic fluid inspired near term by human and rhesus fetuses. *Am J Obstet Gynecol* 1976; 125:306-309.
 45. Katz V, Bowes W. Meconium aspiration syndrome: Reflections on a murky subject. *Am J Obstet Gynecol* 1992;166:171-183.
 46. James A. Resuscitation, stabilization, and transport in perinatology. *Current Opinion in Pediatrics* 1992;5:150-155.
 47. Oriot D, Boussemart T, Cardona J, Wood C, Devictor D, Huault G. *Eur J Pediatr*. 1993;152:781.
 48. Zhu X. Neonatal Resuscitation. *World Health Forum* 1993;14:289-290.
 49. Schoenfeld, P, Baker M. Management of cardiopulmonary and trauma resuscitation in the pediatric emergency department. *Pediatrics* 1993;91:726-729.
 50. Losek J, Bonadio W, Walsh-Kelly C, Hennes H, Smith D, Glaeser P. Prehospital pediatric endotracheal intubation performance review. *Pediatr Emerg Med*. 1989;5:1-4.
 51. Moront M, Eichelberger M. Pediatric Trauma. *Pediatric Annals* 1994;23:186-190.
 52. Ludwig S, Fleisher G. Pediatric cardiopulmonary resuscitation: a review and a proposal. *Pediatr Emerg Care* 1985;1:40-44.
 53. O'Rourke PP. Outcome of children who are apneic and pulseless in the emergency room. *Crit Care Med* 1986;14:466-468.
 54. Sheikh A, Brogan T. Outcome and cost of open and closed-chest cardiopulmonary resuscitation in pediatric cardiac arrests. *Pediatrics* 1994;93:392-398.
 55. Fisher D, Wraape V. Outcome of cardiopulmonary resuscitation in children. *Pediatr Emerg Care* 1987;3:235-238.
 56. Bedell S, Delbanco T, Cook E, Epstein F. Survival after cardiopulmonary resuscitation in the hospital. *N Engl J Med* 1983;309:569-576.
 57. Zaritsky A, Nadkarni V, Getson P. CPR in children. *Ann Emerg Med* 1987;16:1107-1111.
 58. Quan L, Wentz K, Gore E, Copass M. Outcome and predictors of outcome in pediatric submersion victims receiving prehospital care in King County, Washington. *Pediatrics* 1990;86:586-593.
 59. Seidel J. I. Emergency medical services and the pediatric patient: Are the needs being met? II. Training and equipping emergency medical services providers for pediatric emergencies. *Pediatrics* 1986;78:808-812.
 60. Altieri M, Bellet J, Scott H. Preparedness for pediatric emergencies encountered in the practitioner's office. *Pediatrics* 1990;85:710-714.
 61. Inaba A, Seward P. An approach to pediatric trauma. Unique anatomic and pathophysiologic aspects of the pediatric patient. *Emerg Med Clinics of North Am*. 1991;9:523-545.
 62. Cornell H. Evaluation, treatment, and transport of pediatric patients with shock. *Pediatr Clin North Am* 1993;40:303-319.
 63. Rieger A, Berman J, Striebel H. Initial resuscitation and vascular access. *International Anesthesiology Clinics* 1994;32:47-77.
 64. Rosetti J, Thompson B, Arahamian C. Difficulty and delay in intravascular access in pediatric arrest. *Ann Emerg Med* 1984;13:406.
 65. Guy J, Haley K, Zuspan S. Use of intraosseous infusion in the pediatric trauma patient. *J Pediatr Surg* 1993;28:158-161.
 66. Chameides L. CPR challenges in pediatrics. *Ann Emerg Med* 1993;388-392.
 67. Aijian P, Tsai A, Knopp R, Kallens G. Endotracheal intubation of pediatric patients by paramedics. *Ann of Emerg Med*. 1989;18:489-494.
 68. Guss D, Poslusny M. Paramedic orotracheal intubation: A feasibility study. *Am J Emerg Med* 1984;2: 399-401.
 69. Jacobs L, Bernzbeitia L, Bennett B. Endotracheal intubation in the prehospital phase of emergency care. *JAMA* 1983;250:2175-2177.
 70. Nakayama D, Gardner M, Rowe M. Emergency endotracheal intubation in pediatric trauma. *Ann Surg* 1990;211:218-223.

Malinowski Discussion

Hamill: I actually have one question with regard to the children, the submersion victims.¹ I suspect in Seattle they were also hypothermic, if not profoundly hypothermic.

1. Quan L, Wentz K, Gore E, Copass M. Outcome and predictors of outcome in pediatric submersion victims receiving prehospital care in King County, Washington. *Pediatrics* 1990;86:586-593.

Malinowski: They compared the body temperatures of the two groups,

and the group that was, on average, most hypothermic had the poorest outcome, in their analysis.

Pepe: I believe that the reason why the ones who were warmer had the better outcome was that they were the ones who actually went into one of our Seattle lakes, and they were swooped out pretty quickly. The ones who went into the oceanside or the bayside were colder and more difficult to pull out. Another point about kids who do have serious submersions and survive—most of them get resuscitated right away by the bystanders. If you look at

cases where the victims are apparently apneic and cyanotic they are later found to have water in their lungs, we get about 80% of those, receiving immediate bystander CPR, successfully resuscitated. Furthermore, the majority of them will turn out neurologically OK. In contrast, if we get there and if the paramedics have to intubate the victim and continue CPR—or if CPR is still going on when we arrive—we rarely get a survivor. What's interesting, therefore, is that CPR seems to be the key intervention, and it's probably the airway management that turns them around. A lot of these cases may

be 'dry drownings' or, more appropriately, 'near dry drownings'.

Recently we've done a little bit of work on out-of-hospital pediatric arrests and found that nearly 90% of them get successfully intubated. Still, the outcomes are pretty miserable, if we have to initiate or continue CPR for drownings, trauma, whatever it may be. One thing I wanted to ask you, Do you folks still recommend preoxygenation on pediatric patients, or do you go directly to intubation?

Malinowski: Let me just tell you a little bit about the different groups. In the very low birthweight baby, less than 1,000 grams, immediate intubation without delay is preferred. I don't know that the guidelines still suggest it, but I know that it's common practice. In the typical term-newborn, 100% oxygen and ventilation precede intubation. In the pediatric patient, I don't think there are any standards on preoxygenation. The emphasis is on an establishing a patent airway and ventilation. Common sense should prevail, and in a cyanotic, apneic child, a few adequate breaths with 100% oxygen prior to an interruption with an intubation attempt may be warranted.

I did want to comment on your statements about bystander CPR. An important thing to point out is that it's the ventilation that those bystanders are doing that is probably making the most impact.

Pepe: Something that someone had to say yesterday was pretty interesting, regarding the kind of basic CPR that we generally use right now. A lot of people can't understand why CPR is such a success in Seattle but doesn't seem to work elsewhere. I agree that once a patient's gone on about 3-4 minutes or even less, in an arrest, the basic CPR that we use today is probably pretty worthless because the patients are so vasodilated or have so

much vascular collapse because of the anoxia. One of the things that I've seen out-of-hospital and most of us have seen in the hospitals is that if someone arrests in front of us, we intubate and start CPR right away. Then we see the patient begin to have spontaneous eye-opening, move his arms around just with the kind of basic CPR we're using right now. I think that's actually fairly effective, physiologically, if you use it right away. In Seattle, so many people are trained in CPR, that it's usually your family member who does it right away (versus the nurse from across the street or the security guard down the hall, who may perform early CPR but 3-4 minutes out); so, as a result, there is a much higher probability that you're going to get immediate and pretty-good CPR, generally, in Seattle versus other places. So it's a problem when they do correlations using questions like, Did the patient get CPR? I think that's a misleading or inadequate data point because it's actually more of an issue to determine the time that the patient got the CPR.

Kaye: I think you did a marvelous job, and, indeed, you kept me awake, because I know little about children and absolutely nothing about neonates. I was intrigued that it's not the ABCs, it's the DWSs, right? Dry, warm, and stimulate. You have to learn your DWSs—I like that. By the way, we all believed in the ACLS program 20 years ago, and we still believe in it. Don't misunderstand me. Actually, the new ACLS course is heavily based on the PALS program. The PALS people had the opportunity to look at the standard ACLS program and actually create a better program.

Malinowski: I think there were more educators involved right at the start of the PALS and the NRP—people who were specialists in education.

Kaye: That's important.

Rubenfeld: I appreciated your introductory comment, "I realize now, that adults are not just big neonates!" and as somebody who takes care of "big neonates," I have a couple of comments. I recently took the PALS and NRP courses. Not being a parent, the thing that I found most difficult about it and that was different from ACLS is that ACLS is very heart rate and rhythm oriented. A lot of PALS and NRP material seems to be focused on how the kid looks, which is something that nurses and physicians who take care of a lot of kids know innately. For people like me, it's completely foreign and when you mentioned it, I was just wondering whether educators who are involved in PALS training have thought about more creative ways, perhaps using videotapes or other teaching methods, to teach big-neonate doctors like me what a sick kid looks like—what a sick 1-month-old looks like, what a sick 2-year-old looks like, things like that.

Malinowski: No, I don't have a good answer for that question, because I know that we haven't personally looked at any way of trying to get that information across. It's like any clinical practice—some of it just takes experience, but I think that if the PALS and the NRP course educators start to do more scenario-based teaching, like ACLS, that might help—just using examples of what an 8-month-old should look like. Because I am a parent, it's become much easier for me, because of my experience of what a 2-year-old should do and look like. But it might be that we need more reinforcing of that and we need to discuss those things, especially because, as I said, there is such a great impact with early intervention. You're working with individuals who have a lot of potential for future health and for good quality of life.

Manuscript-Preparation Instructions for Authors and Typists

General Information

RESPIRATORY CARE welcomes original manuscripts related to respiratory care and prepared according to these Instructions. Perfection is not required, but efforts in that direction are appreciated. **Computer diskette** submissions are encouraged and may reduce processing and review time. See requirements in these Instructions.

Editorial consultation is available by telephone or letter at any stage of planning or writing. Specific guidance (in printed form) will be provided on request for writing a research paper, a case report, an evaluation, a review, overview, or update or a book review; for converting to and from SI units; and for in-house manuscript review. For typists, a model manuscript, list of journal name abbreviations, and copy of these Instructions is available. Write to RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229-4593, call (214) 243-2272, or fax (214) 484-6010.

Manuscripts are **reviewed** by authoritative referees in a double-blind manner. Accepted manuscripts may be copyedited for clarity and style; authors receive **galley**s to proofread before publication. Published papers are **copyrighted** by the publisher and may not be published elsewhere without permission.

Publication Categories

Research Article: A report of an original investigation (a study).

Evaluation of Device/Method/Technique: A description and evaluation of an old or new device, method, technique, or modification.

Case Report: A report of a clinical case that is uncommon, or was treated in a new way, or is exceptionally instructive. All authors must have been associated with the case. A case-managing physician must either be an author or furnish a letter approving the manuscript.

Review Article: A comprehensive, critical review of the literature and state-of-the-art summary of a pertinent topic that has been the subject of at least 40 published research articles.

Overview: A critical review of a pertinent topic about which not enough has been published to merit a Review Article.

Update: A report of subsequent developments in a topic that has been critically reviewed in this journal or elsewhere.

Point of View Paper: A paper expressing personal but substantiated opinions on a pertinent and controversial topic.

Special Article: A pertinent paper not fitting one of the foregoing categories may be acceptable as a Special Article. It is advis-

able to consult the Editor before writing or submitting such a paper.

Editorial: A paper drawing attention to a pertinent concern; it may present an opposing opinion, clarify a position, or bring a problem into focus.

Letter: A signed communication about prior publications in this journal or about other pertinent topics. Tables and illustrations may be included. Type double-spaced, supply a title, mark "For publication."

Blood Gas Corner: A brief, instructive case report involving respiratory care blood data—with questions, answers, discussion.

PFT Corner: Like Blood Gas Corner, but involving pulmonary function tests.

Test Your Radiologic Skill: Like Blood Gas Corner, but involving pulmonary medicine radiography and including one or more radiographs, may involve imaging techniques other than conventional chest radiography.

Review of Book, Film, Tape, or Software: A balanced, critical review of a recent release.

Considerations

Prior and Duplicate Publication: Work that has been *published* or *accepted* elsewhere usually should not be submitted. In special instances, the Editor may consider such material, provided that permission to publish is given by the author and other publisher. Please consult the Editor before submitting such work.

Authorship: All persons listed as authors should have participated in the reported work and the shaping of the manuscript; all should have proofread the submitted manuscript; and all should be able to publicly discuss and defend the paper's content. A paper with collective (corporate) authorship must specify the key persons responsible for the article. Authorship is not justified solely on the basis of solicitation of funding, collection or analysis of data, provision of advice, or similar services. Persons performing such ancillary services may be recognized in the Acknowledgments section.

Conflict of Interest: Authors of research or evaluation papers, points of view, or editorial are asked to disclose on the manuscript's title page any liaison or financial arrangement they may have with a manufacturer or distributor whose product figures in the submitted manuscript or with the manufacturer or distributor of a competing product. (Such arrangements will not disqualify a paper from consideration and will not be disclosed to reviewers.)

Preparation of the Manuscript

Note: in addition to reading these Instructions, authors and typists can benefit from inspecting papers recently published in *RESPIRATORY CARE* and using them as models.

General Specifications

Type on one side of white bond paper, 216 × 279 mm (8 in. × 11 in.) with margins of at least 25 mm (1 in.) on all sides of the page. **Double-space** the entire manuscript (three lines per vertical inch). Number all pages in upper-right corners. Indent paragraphs 5 spaces. Do **not justify**. Do **not** underline titles, headings, or other words. Do **not** type authors' names or other identification anywhere except on the title page. Repeat title only (no authors) on the abstract page. Begin each of the following on a new page: title page, abstract, text, product-sources list, acknowledgments, reference list, each table, each appendix, list of figure captions. Use standard English. Employ the first person and active voice (eg, "We believe that pigs can fly") rather than the 'obscure person' and passive voice (eg, "It is believed that pigs can fly")—because the latter obscures the identity of the responsible party (the believer).

Headings in Text: Center *main section headings* on the page and type them in capital and small letters (eg, Introduction, Methods, Results, Discussion). Begin *subheadings* at the left margin and type them in capital and small letters (eg, Patients, Equipment, Statistical Analysis). Do **not underline or darken** section headings or subheadings.

Manuscript Structure

Most kinds of papers have standard parts in a standard order, as shown hereafter. However, papers can vary individually, and not all papers will have all the parts listed here.

Research Article: Title Page, Abstract, Introduction, Methods, Results, Discussion, Conclusions, Product Sources, Acknowledgments, References, Tables, Appendices, Figure Legends.

Evaluation of Device/Method/Technique: Title Page, Abstract, Introduction, Description of Device/Method/Technique, Evaluation Methods, Evaluation Results, Discussion, Conclusions, Product Sources, Acknowledgments, References, Tables, Appendices, Figure Legends.

Case Report: Title Page, Introduction, Case Summary, Discussion, References, Tables, Figure Legends.

Review Article: Title Page, Table of Contents, Introduction, Review of the Literature, State-of-the-Art Summary, Acknowledgments, References, Tables, appendices, and illustrations may be included. Other formats may be suitable.

Point of View Paper: Title Page, Text, References. Tables and illustrations may be included.

Details about Sections:

Title: Make the paper's title as specific, clear, and yet as short as you can.

Title Page: List (a) title of the paper; (b) full names of all authors, with academic and credential letters, professional titles, and institutional affiliations; (c) name, address (include building and/or room number for courier service), telephone number, and Fax number of corresponding author; (d) name and address for reprint requests; (e) sources of support such as grants, equipment, drugs, and supplies; (f) name of organization, location, and date of any meeting at which a version of the paper has been presented; (g) disclosure of financial relations of any author with commercial products or interests connected with the paper—or with competing products or interests; (h) name, title, and affiliation of statistical consultant, if any; and (i) disclaimers, if any.

Abstract: (required only for research articles and evaluations of devices/ methods/techniques). The abstract must summarize what was studied; why and how it was studied; the results, including important data and statistical significance; and conclusions drawn from the results. All information in the abstract must also appear in the paper itself. Do not cite references in the abstract. The abstract for a research article should include the following headings (in all capital letters), appropriately placed within the abstract and followed by colons: **BACKGROUND, METHODS, RESULTS, CONCLUSIONS**. The abstract for a paper evaluating a device/method/technique should include the following headings: **BACKGROUND, DESCRIPTION OF DEVICE, EVALUATION METHODS, EVALUATION RESULTS, CONCLUSIONS**. The abstract should be all one paragraph, not indented, and not longer than 250 words. Center title, typed in capital and lower case letters, over abstract.

Introduction: Briefly describe the background of the work or the paper. Cite only pertinent references, and do not review the subject extensively. Do not include data or conclusions from the work reported in your paper. In a research paper, end this section with a clear statement of the research question(s) or hypothesis(es).

Methods Section (in a research paper): Describe the selection of patients, controls, or laboratory animals. Give details about randomization. Describe methods for blinding of observations. Give numbers of observations. Report losses to observation (eg, dropouts or disqualified subjects), listing numbers of subjects or data sets lost, when lost, and why lost. *Describe methods in sufficient detail to allow other workers to replicate your work.* Give references to established methods; provide references and brief descriptions for methods that have been published but are not well known; describe new or substantially modified methods, give reasons for using them, and evaluate their limitations. Report calibration of measuring devices.

Drugs—Identify precisely all drugs and chemicals used, giving generic names, doses, and routes of administration. If desired, brand names may be given in parentheses after generic names.

Commercial Products—Identify any commercial product (including model number if applicable) the first time it is mentioned, giving the manufacturer's name, city, and state or country—in parentheses in the text. If four or more products are mentioned, do not list any manufacturers in the text; instead, list them on a Product Sources page at the end of the text before the References. Provide model numbers when available and manufacturer's suggested price if the study has cost implications.

Ethics—When reporting experiments on human subjects, indicate that procedures were in accordance with the ethical standards of the institution's committee on human experimentation. State that informed consent was obtained after the nature of the procedure(s) had been explained. Do not use patient's names, initials, or hospital numbers in text or illustrations. When reporting experiments on animals, indicate that the institution's or any national guide or national law on the care and use of laboratory animals was followed.

Statistics—In the last paragraph of the Methods section, identify the statistical tests used in analyzing the data, and give the prospectively determined level of significance. Cite references to support choices of tests. (Cite textbooks or published articles, not handbooks of commercial software.) Identify any general-use or commercial computer programs used, naming manufacturers and their locations.

Results Section: Present results in logical sequence in the text. Tables and illustrations may also present data. Do not repeat in the text all the data in the tables or illustrations; emphasize or summarize only important observations and trends. Be sure to report all the results; do not save some of them for the Discussion section. Do not discuss the findings in the Results section. Exact *p* values are preferred in all cases but are *essential* when values are not statistically significant. Do not report original results merely as nonsignificant or NS.

Discussion Section: It may be useful to restate the research question(s), but do not repeat in detail the data or other material given in the Introduction, Methods, or Results sections. Emphasize the new and important aspects of the study and the conclusions that follow from them. Present the implications and limitations of the findings—including implications for future research. Relate the findings to other relevant published work. Link the conclusions with the goals of your work, but avoid unqualified statements and conclusions not completely supported by your data. Avoid claiming priority and alluding to work that has not been completed. State new hypotheses when warranted, but clearly label them as such. Recommendations, when appropriate, may be included. Provide a clear 'take-away' message for readers—either at the end of the Discussion section or in a separate **Conclusions** section.

Product Sources Page: When four or more commercial products, including statistical software, are mentioned in the paper, list manufacturers' names, cities, and states or countries on a Product Sources page after the text. For each kind of product, list the generic term, brand name and model number, manufacturer's name, city, and state

or country. Manufacturer's suggested price should be included when the study or evaluation has cost implications. For example:

Manual Resuscitators:

BagEasy, Respironics Inc, Murrysburg PA, \$20.50

Code Blue, Vital Signs Inc, Totowa NJ, \$19.85

Ventilators:

7200, Puritan-Bennett Corp, Overland Park KS

Bear Cub, Bear Medical Systems, Riverside CA

Acknowledgments Page: On this page you may recognize the services of persons who made ancillary contributions to the work or the manuscript. Such services might be advice about methodology; data collection; statistical advice or analysis; equipment selection or operation; cooperation as caregiver, patient, or subject; manuscript preparation; in-house review; and other services. Each acknowledgment must specify the service rendered. Named persons must provide written agreement (accompanying submitted manuscript) to be so recognized.

References

Use of References: References are used to support statements of fact, to indicate sources of information, or to guide readers to further information. Be careful to make clear in the text the reason for a specific citation (ie, do not imply support of a statement of fact by citing a reference that simply addresses the issue). Cite only sources that have actually been consulted and evaluated by the authors. Cite only *published* or *accepted* material. Cite original articles in preference to textbooks, review articles, abstracts, editorials, or letters. Avoid citing abstracts because some published abstracts have never been subjected to peer review and may not support your contention. Make every effort to determine whether an abstract has been subsequently published as a full-length paper. Avoid citing non-English language sources. When citing from a book, specify the page numbers unless you are citing the entire book. If you cite a paper that has been accepted but not yet published ("in press"), provide a copy of the paper to the Editor when you submit your manuscript.

Do not cite unpublished observations as references. Instead, identify written (not oral) communications in parentheses in the text, giving the writer's name and location and the date of the communication. Information from manuscripts submitted but not yet accepted should be cited in the text (in parentheses) as "unpublished observations."

Citing References in the Text: The first reference you cite is Reference 1, the next is Reference 2, etc. After the first citation of a reference, use its original number if you cite it again later in the paper. Cite references by superscript, full-size, arabic numerals. **Do not enclose in parentheses.** If a citation numeral is located at the end of a phrase or sentence, place the numeral after (outside) the comma, semicolon, or period—not before (inside) it. Avoid citing references at the end of a phrase or sentence if they pertain only to internal parts of the phrase or sentence; instead, cite them at the pertinent places within the phrase or sentence (eg, Hess et al¹³ discovered that ...").

Listing References: Starting on a new page after the text, list the references in numerical order. Do not employ "op cit" or "ibid." Type references double-spaced, using the styles of the examples given hereafter. List all authors (do not use "et al"). In titles of articles and books, capitalize only first words and proper names. Abbreviate journal names as in Index Medicus. Spell out in full the names of less well known or nonindexed journals and periodicals. If the cited item is an abstract, editorial, or letter, identify it as such in parentheses following the item's title. Provide both first and last complete page numbers. Do not leave spaces between dates and volume and page numbers. Obtain authors' names, article and book titles, dates, and volume and page numbers from the *original* cited articles and books, not from other articles' reference lists, which often are inaccurate. Examples of correct reference listings follow (these are single-spaced here but must be *double-spaced* in a manuscript).

Article in a journal carrying pagination throughout volume:

1. Shepherd KE, Johnson DE. Bronchodilator testing: an analysis of paradoxical responses. *Respir Care* 1988; 33:667-671.

Article in publication that numbers every issue beginning with Page 1:

2. Bunch D. Establishing a national database for home care. *AARC Times* 1991;15(Mar):61,62,64.

Corporate author journal article:

3. American Association for Respiratory Care. Criteria for establishing units for chronic ventilator-dependent patients in hospitals. *Respir Care* 1988;33:1044-1046.

Article in journal supplement: (Journals differ in their methods of numbering and identifying supplements. Supply sufficient information to allow retrieval.)

4. Reynolds HY. Idiopathic interstitial pulmonary fibrosis. *Chest* 1986;89(3, Suppl):139s-143s.

Abstract in journal: (Abstracts are not strong references. Abstracts more than 3 years old should not be cited. When cited, abstracts should be identified as such.)

5. Stevens DP. Scavenging ribavirin from an oxygen hood to reduce environmental exposure (abstract). *Respir Care* 1990;35:1087-1088.

Editorial in journal:

6. Rochester DF. Does respiratory muscle rest relieve fatigue or incipient fatigue? (editorial). *Am Rev Respir Dis* 1988;138:516-517.

Editorial with no author given:

7. High frequency ventilation (editorial). *Lancet* 1991; 1:706-708.

Letter in journal:

8. Smith DE, Herd D, Gazzard BG. Reversible bronchoconstriction with nebulised pentamidine (letter). *Lancet* 1988;2:905.

Paper accepted but not yet published:

9. Hess D. New therapies for asthma. *Respir Care* (year, in press).

Personal author book: (Specific pages should be cited whenever possible.)

10. Nunn JF. Applied respiratory physiology. New York: Appleton-Century Crofts, 1969.

Note: To specify pages in a book, place a colon after the year and then list the page(s). Examples: 1969:85 (one page), 1969:85-95 (series of contiguous pages), 1969:85,95 (separated pages).

Corporate author book: (Specific pages should be cited whenever possible.)

11. American Medical Association Department of Drugs. AMA drug evaluations, 3rd ed. Littleton CO: Publishing Sciences Group, 1977.

Book with editor(s): (Specific pages should be cited whenever possible.)

12. Guenter CA, Welch MH, editors. Pulmonary medicine. Philadelphia: JB Lippincott, 1977.

Chapter in book: (Specific pages should be cited whenever possible.)

13. Pierce AK. Acute respiratory failure. In: Guenter CA, Welch MH, editors. Pulmonary medicine. Philadelphia: JB Lippincott, 1977:171-223.

Newspaper article:

14. Rensberger B, Specter B. CFCs may be destroyed by natural process. *The Washington Post* 1988 Aug 7;Sect A:2(Col 5).

Dictionary or similar reference:

15. Pneumohemopericardium. Dorland's illustrated medical dictionary, 26th ed. Philadelphia: WB Saunders, 1981: 1038.

Tables: Use tables to display information, compare data, or show trends. Start each table on a separate page. Do not construct a table with fewer than two lines (rows) or columns of data (instead, put the data in the text). Avoid more than 8 columns across. Number tables as Table 1, Table 2, etc, consecutively in the order of their first mention in the text. Place the number and a **descriptive** title above the table (not on a separate page). Give each column a brief heading. Place explanatory matter in footnotes, not in the title or column headings. Explain in footnotes all nonstandard abbreviations and symbols used in the table. To key footnotes to the table body, use conventional designations (aster-

isk, dagger, double dagger, etc) in consistent order, placing them superscript in the table body.

Double-space all elements of tables, including titles, column headings, data, and footnotes. Continue a deep table on following pages. Do not use horizontal or vertical rules. Do not submit tables as photographs, or reduced in size, or on oversize paper. Use the same typeface as in the text. Supply the name and version of any table-building computer program used.

Appendices: Mathematical calculations, documents, and other matter that would clutter the main article can be displayed in appendices. Number them as Appendix 1, Appendix 2, etc., and refer to them in the text. Give each appendix a descriptive title and type it double-spaced throughout.

Illustrations: Graphs, line drawings, photographs, and radiographs are called figures. Use only illustrations that clarify and augment the text. Number them consecutively as Fig. 1, Fig. 2, etc., according to the order in which they are first mentioned in the text. Figures for publication must be of professional quality, but rough sketches may accompany the submitted manuscript, with final figures to be prepared after review. Figures need not be photographic reproductions. Clear, clean laser-printer-generated figures are acceptable (121-144 dpi). However, the data from which the original graphs are generated should be available to the editor upon request. Remember that originals that are roughly 7 5/9 inches will be reduced to less than 50% (3 5/4 in) and originals with a horizontal dimension of 9 in will be reduced to less than 33%. Photographs must be glossy 5 5/7 to 8 5/10-inch black and white prints, unless color is essential. (If color is essential, consult the Editor to learn whether negatives, transparencies, or prints are required.) In reports of animal experiments, use schematic drawings, not photographs. A letter of consent must accompany any photograph in which a possibility of identification of a person exists; masking the eyes is not sufficient. Lettering and numerals must be neat, uniform in size and style, and large enough to remain legible when downsized for publication. Do not place titles and detailed explanations on figures; put such information in the figure captions. Identify each figure on the back with a stick-on label showing figure number, an arrow indicating the top, and an abbreviated manuscript title. *Omit author's name.* Cover label with clear tape so ink will not smudge other prints. Do not use staples or paper clips, and do not write heavily on the backs of prints.

Radiographs: If possible, submit radiographs as full-size copies of films, not as prints. Prints may be acceptable, but full-size films are preferable in order to display better detail in published figures. Be sure all figures are cited in the text. If any figure has been published before, include copyright-holder's written permission to use it.

Figure Captions: Its caption should, to the extent possible, make a figure understandable without referring the reader to the text. Type figure captions double-spaced, on a separate page, as Fig. 1, Fig. 2, etc. When symbols, arrows, numbers, or letters are used to identify parts of a figure, identify and explain each part clearly in the caption. In photomicrographs, explain the internal scale and method of staining. If a figure has been published

before, acknowledge the original source in its caption (permission must be obtained prior to use, of course).

Units of Measurement: Give measurements of length, height, weight, and volume in metric units appropriately abbreviated. Give temperatures in degrees Celsius. Give blood pressures in millimeters of mercury (mm Hg). Report hematologic and clinical-chemistry measurements in conventional metric system and in SI units (International System of Units). Show gas pressures (including blood gas tensions) in torr. List SI equivalent values, when possible, in brackets following non-SI values—for example, "PEEP, 10 cm H₂O [0.981 kPa]." For conversion to SI, see *RESPIRATORY CARE* 1988;33:861-873 (Oct 1988) and 1989;34:145 (Feb 1989).

Arithmetic: Carefully double-check all arithmetic before submitting the paper. Accuracy is the author's responsibility; errors are common!

Abbreviations and Symbols: Use standard abbreviations and symbols. Avoid creating new abbreviations. Avoid all abbreviations in the title and unusual abbreviations in the abstract. Use an abbreviation only if the term occurs several times in the paper. Write out the full term the first time it appears, followed by the abbreviation in parentheses. Thereafter, employ the abbreviation alone. Never use an abbreviation without defining it. Standard units of measurement can be abbreviated without explanation (eg, 10 L/min, 15 torr, 2.3 kPa). If you employ a great many abbreviations and symbols, provide a double-spaced list of them, with their definitions, in alphabetical order.

Please use the following forms: cm H₂O (not cmH₂O), f (not bpm), L (not l), L/min (not LPM, l/min, or lpm), mL (not ml), mm Hg (not mmHg), pH (not Ph or PH), $p > 0.001$ (not $p > 0.001$), s (not sec), SpO₂ (pulse oximetry saturation).

Computer Diskettes: A manuscript may be submitted on a Macintosh or IBM-compatible diskette. Macintosh documents on 3.5 in. diskettes written in Microsoft Word versions 4.0 and 5.0 are preferred. However, we can convert most documents from software (including PC-DOS format) to a Macintosh-Microsoft Word document.

Label each diskette with date; author's name; name of word-processing program and version used to prepare documents; and filename(s). If not enough space is available, list contents on disk jacket or an attached note. Do not write on a diskette except with a felt-tipped pen.

Tables and figures must be in their own separate files, with software identified.

Together with diskette, supply three hard copies of the manuscript. Do not paperclip a diskette to its hard copy.

Proofreading and In-House Review: Have all authors proofread the manuscript for content accuracy and language. Consider having the manuscript reviewed in-house by colleagues before submitting it.

Submitting the Manuscript

Use the checklist below to make sure the manuscript is ready for mailing. Mail three copies of the manuscript and figures to RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229-4593. Do not Fax manuscripts. Protect figures with cardboard to prevent bending. A computer diskette submission must be accompanied by the requisite three hard copies. Keep a copy of the manuscript and figures in your files in case of loss. You will be sent an acknowledgment that your manuscript has been received.

Cover Letter: The manuscript must be accompanied by a covering letter signed by all the authors. The letter must specify the intended publication category and, when there are two or more authors, state that "We, the undersigned, have all participated in the work reported, proofread the accompanying manuscript, and approved its submission for publication."

Permissions: The manuscript must be accompanied by copies of permissions to reproduce published material (figures or tables); to use illustrations of, or report sensitive personal information

about, identifiable persons; or to name persons in the Acknowledgments section.

Author's Checklist:

1. Does paper fit a listed publication category?
2. Does the cover letter meet specifications?
3. Is the title page complete?
4. Is double-spacing used throughout entire manuscript?
5. Are all pages numbered in upper-right corners?
6. Are paragraphs indented 5 spaces?
7. Are all references, figures, and tables cited in the text?
8. Are references typed in requested style?
9. Have SI values been provided?
10. Has all arithmetic been checked?
11. Have generic names of drugs been provided?
12. Have necessary written permissions been provided?
13. Have authors' names been omitted from text and figure labels?
14. Have copies of "in press" references been provided?
15. Has manuscript been proofread by all authors?

JUNE 30 DEADLINE FOR AARC FELLOWSHIPS

The Allen and Hanburys Fellowship for Asthma Education provides \$3,500 to allow completion of a project encompassing asthma self-management or asthma awareness. The Fellowship also provides airfare and one night's lodging to attend the Awards Ceremony at the AARC Annual Convention.

Three \$1,000 fellowships are available. In addition to the cash award, each includes airfare and one night's lodging to attend the Awards Ceremony at the AARC Annual Convention.

- **The Lifecare Fellowship** is designed to foster projects dealing with mechanical ventilation, especially out side of the ICU.
- **The Monaghan/Trudell Fellowship** is designed to support projects dealing with the development of cost-effective aerosol delivery.
- **The Respironics Fellowship** is designed to foster projects dealing with noninvasive ventilatory support.

For details and specifications for application, contact Lynn Perkins or Joy Rea in the AARC Executive Office by phone (214) 243-2272 or FAX (214) 484-2720.

AARC SUMMER FORUM
Vail, Colorado July 14-16, 1995

RESPIRATORY CARE WEEK
October 1-7, 1995

LAST CALL FOR OPEN FORUM 1995

It's time to submit your abstracts for possible presentation at the OPEN FORUM during the AARC Annual Meeting in Orlando, Florida, December 2-5, 1995. For information on changes to the rules/instructions, see Page 595 of this issue. **Final deadline, May 27, 1995 (postmark).**

THE NATIONAL BOARD FOR RESPIRATORY CARE—1995 Examination and Fee Schedule

	Examination Date	Application Deadline		Examination Date	Application Deadline
CRTT:	July 15, 1995 November 11, 1995	May 1, 1995 September 1, 1995	CPFT:	June 3, 1995	April 1, 1995
RRT:	June 3, 1995 December 2, 1995	February 1, 1995 August 1, 1995	RPFT:	December 2, 1995	September 1, 1995

Fee Schedule

				Active	Inactive
CRTT					
—new applicant:	\$ 90.00		CRTT Recredentialing:	\$25.00	\$ 60.00
—reapplicant:	\$ 60.00		RRT Recredentialing:		
RRT Written and Clinical Simulation			Written Registry Examination	\$25.00	\$ 60.00
—new applicant:	\$190.00		Clinical Simulation Examination	\$65.00	\$100.00
—reapplicant:	\$160.00		CPFT Recredentialing:	\$25.00	\$ 80.00
Written Registry Only			RPFT Recredentialing:	\$25.00	\$130.00
—new applicant:	\$ 90.00		P/P Specialty Credentialing:	\$25.00	\$130.00
—reapplicant:	\$ 60.00		Membership Renewal:		
Clinical Simulation Only—new & reapplicant:	\$100.00		CRTT/RRT/CPFT/RPFT		\$ 17.00
CPFT					
—new applicant:	\$100.00		Credential Verification	\$ 2.00	\$ 15.00
—reapplicant:	\$ 80.00		Replacement Certificate	\$ 6.00	\$ 25.00
RPFT			Copy of NBRC Directory: 1994		
—new applicant:	\$150.00		Active Credentialed Practitioners	N/C	\$ 25.00
—reapplicant:	\$130.00		Copy of NBRC 1994 Listing of All		
Perinatal/Pediatric Specialty			Credentialed Practitioners	\$10.00	\$ 25.00
—new applicant:	\$150.00				
—reapplicant:	\$130.00				

8310 Neiman Road • Lenexa, Kansas 66214 • (913) 599-4200

1995 Call for Abstracts

RESPIRATORY CARE • OPEN FORUM

The American Association for Respiratory Care and its science journal, *RESPIRATORY CARE*, invite submission of brief abstracts related to any aspect of cardiorespiratory care. The abstracts will be reviewed, and selected authors will be invited to present papers at the OPEN FORUM during the AARC Annual Meeting in Orlando, Florida, December 2-5, 1995. Accepted abstracts will be published in the November 1995 issue of *RESPIRATORY CARE*. Membership in the AARC is not necessary for participation.

SPECIFICATIONS—READ CAREFULLY!

An abstract may report (1) **an original study**, (2) **the evaluation of a method or device**, or (3) **a case or case series**. Topics may be aspects of adult acute care, continuing care/rehabilitation, perinatology/pediatrics, cardiopulmonary technology, health-care delivery. The abstract may have been presented previously at a local or regional—but not national—meeting and should not have been published previously in a national journal. The abstract will be the only evidence by which the reviewers can decide whether the author should be invited to present a paper at the OPEN FORUM. Therefore, *the abstract must provide all important data, findings, and conclusions*. Give specific information. Do not write such general statements as “Results will be presented” or “Significance will be discussed.”

Essential Content Elements

An original study abstract *must* include (1) Introduction: statement of research problem, question, or hypothesis; (2) Method: description of research design and conduct in sufficient detail to permit judgment of validity; (3) Results: statement of research findings with quantitative data and statistical analysis; (4) Conclusions: interpretation of the meaning of the results. **A method/device evaluation** abstract *must* include (1) Introduction: identification of the method or device and its intended function; (2) Method: description of the evaluation in sufficient detail to permit judgment of its objectivity and validity; (3) Results: findings of the evaluation; (4) Experience: summary of the author's practical experience or a lack of experience; (5) Conclusions: interpretation of the evaluation and experience. Cost comparisons should be included where possible and appropriate. **A case report** abstract *must* report a case that is uncommon or of exceptional teaching/learning value and must include (1) patient data and case summary and (2) significance of case. Content should reflect results of literature review. The author(s) should have been actively involved in the case and a case-managing physician must be a co-author or must approve the report.

Abstract Format and Typing Instructions

Accepted abstracts will be photographed. First line of abstract should be the title in all capital letters. Title should explain content. Follow title with names of all authors (including credentials), institutions(s), and location. Underline presenter's name. Type or electronically print the abstract *single spaced in the space provided on the abstract blank*. Insert only one letter space between sentences. Text submission on diskette is encouraged but must be accompanied by a hard copy. Identifiers will be masked (blinded) for review. Make the abstract all one paragraph. Data may be submitted in table form and *simple figures may be included provided they fit within the space allotted*. No figures, illustrations, or tables are to be attached to the abstract form. Provide all author information requested at the bottom of abstract form. A clear photocopy of the abstract may be used. Standard abbreviations may be employed without explanation. A new or infrequently used abbreviation should be preceded by the spelled-out term the first time it is used. Any recurring phrase or expression may be abbreviated if it is first explained. Check the abstract for (1) errors in spelling, grammar, facts, and figures; (2) clarity of language; and (3) conformance to these specifications. An abstract not prepared as requested may not be reviewed. Questions about abstract preparation may be telephoned to the editorial staff of *RESPIRATORY CARE* at (214) 243-2272.

Deadline Allowing Revision

Authors may choose to submit abstracts early. Abstracts received by March 15 will be reviewed and the authors notified by April 22. Rejected abstracts will be accompanied by a written critique that should in many cases enable authors to revise their abstracts and resubmit them by the final deadline (May 27).

Final Deadline

The mandatory Final Deadline is May 27 (postmark). Authors will be notified of acceptance or rejection *by letter only*—to be mailed by August 15.

Mailing Instructions

Mail (Do not fax!) 2 clear copies of the completed abstract form and a stamped, self-addressed postcard (for notice of receipt) to:

RESPIRATORY CARE OPEN FORUM
11030 Ables Lane
Dallas, TX 75229-4593

1995 RESPIRATORY CARE OPEN FORUM

Abstract Form

1. Title must be in all upper case (capital) letters, authors' full names and text in upper and lower case.
2. Follow title with all authors' names including credentials (underline presenter's name), institution, and location.
3. Do not justify (ie, do leave 'ragged' right margin).
4. **Do not use type size less than 10 points.** (See reduction samples below.)
5. All text, tables, and figures must fit into the rectangle shown.
6. Submit 2 clean copies. This form may be photocopied if multiple abstracts are to be submitted.

Mail original &
1 photocopy
(along with postage-paid
postcard) to

**RESPIRATORY CARE
OPEN FORUM
11030 Ables Lane
Dallas TX 75229-4593**

*Early deadline is
March 15, 1995
(abstract received)*

*Final deadline is
May 27, 1995
(abstract postmarked)*

13.9 cm or 5.5"

Presenter's Name & Credentials

Presenter's Mailing Address

Presenter's Voice Phone & Fax

Corresponding Author's Name & Credentials

Corresponding Author's Mailing Address

Corresponding Author's Voice Phone & Fax

This is 14-point type, with
necessary reduction.

This is 12-point type, with
necessary reduction.

This is 10-point type,
with necessary reduction.

This is 9-point type,
with necessary reduction
(too small!)

18.8 cm or 7.4"

AARC & AFFILIATES

May 23–25 in Kalispell, Montana. The MSRC presents the 21st Annual Conference of Respiratory Care at Cavanaugh's Inn. Topics include current trends in respiratory care, new approaches to asthma, a sleep disorders update, and new developments in cystic fibrosis. Participants can earn up to 15 MSRC-approved Category I continuing education units; CRCE credit has been requested. For registration information, contact Dave Bunkers at (406) 657-4075; for hotel reservations, call Cavanaugh's Inn at (406) 752-6660.

May 24–26 in Tucson, Arizona. The ASRC presents its annual state seminar, which focuses on both the clinical and the general job market for the respiratory care profession. The seminar, entitled "Our Future Is Now—A Brighter Tomorrow," features AARC Director of Government Affairs Cheryl Brown and Senator Jim Kolbe (R-AZ). Contact Leanna Konechne at (602) 326-1600.

June 7–9 in Round Top, New York. The New Jersey Society for Respiratory Care announces the 8th Annual Managers/Educators Rocking Chair Conference at the Winter Clove Inn and Conference Center. Topics include hospital restructuring, patient-focus care models, program accreditation options, and national and regional updates. The conference is approved for 8 CRCE Category I credit hours. The registration fee of \$225 includes conference registration, lodging for two nights, meals, and gratuities. Contact Ken Wyka at (201) 982-7228 or Joseph Sorbello at (315) 464-6872.

June 7–9 in Henrietta, Oklahoma. The OSRC hosts its annual educational seminar at the Lake Eufala Fountainhead Resort; "Making All the Pieces Fit?" is this year's theme. CRCE credit has been requested. Contact Gennie Ridlen or Cynthia Gray, Hillcrest Health Center, 2129 SW 59th St, Oklahoma City OK 73119 (405) 680-2116.

July 14–16 in Vail, Colorado. The AARC presents the Summer Forum, with emphasis on management and education topics. Consult the April issue of AARC Times for program details and registration information.

June 14–16 in St. Charles, Illinois. The ISRC hosts its 27th Annual State Convention, featuring workshops that examine respiratory protocols, point-of-care blood gas testing, essential assessment skills, and how to improve your professional image. Contact Doug McQueary at (312) 962-4060 or Jane Reynolds at (312) 883-6535.

June 23–24 in Atlanta, Georgia. The GSRC and the Georgia Thoracic Society present a professional education confer-

ence, in conjunction with the American Lung Association of Georgia, at the Ritz Carlton Hotel in Buckhead. Topics include pulmonary vascular disease and reorganization of respiratory care. Contact Lynda Thomas Goodfellow or Vijay Deshpande at Georgia State University, Department of Cardiopulmonary Care Sciences, University Plaza, Atlanta GA 30303, (404) 651-3037, fax (404) 651-1531.

August 23–25 in Albuquerque, New Mexico. The NMSRC announces its Annual Summer Convention at the Albuquerque Convention Center. The program features an I.V. class and a bronchoalveolar lavage class; other topics include postacute care and pressure-control ventilation. Contact Brooke Patterson of THC Hospital at (505) 242-4444.

OTHER MEETINGS

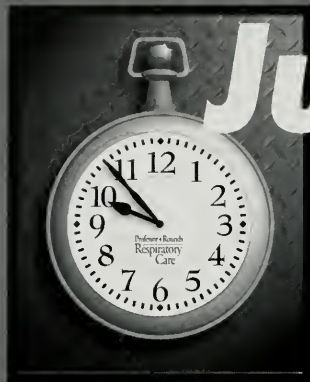
May 20–24 in Seattle, Washington. The American Thoracic Society hosts its annual International Conference, featuring a variety of symposia and workshops on the prevention and control of lung disease. Contact 1995 International Conference, American Thoracic Society, 1740 Broadway, New York NY 10019-4374 (212) 315-8700.

May 30–June 4 in Nashville, Tennessee. The American Sleep Disorders Association and the Sleep Research Society announce their 9th Annual Meeting at the Opryland Hotel. For registration materials and a copy of the program, contact the Annual Meeting Office at (507) 287-6006, x 661 or fax (507) 287-6008.

June 16–19 in Toronto, Ontario, Canada. The Canadian Society of Respiratory Therapists (CSRT) announces its 30th Educational Forum at the Royal York Hotel. Workshop topics cover management, ventilation, cardiopulmonary diagnostics, lung transplantation, and research. Call (416) 368-2511 or fax (416) 368-2884 for more information.

October 12–15 in Minneapolis, Minnesota. The American Association of Cardiovascular and Pulmonary Rehabilitation hosts its 10th Annual Meeting, "Building on Success—A Decade of Progress," at the Minneapolis Convention Center. For registration and hotel information, contact Michele Johnson (608) 831-6989.

October 29–November 2 in New York, New York. The 61st Annual International Scientific Assembly sponsored by the American College of Chest Physicians (ACCP) at the New York Hilton and Towers is entitled "Chest 1995: Prevention and Diagnosis of Chest Disease." Contact the ACCP, 3300 Dundee Rd, Northbrook IL 60062-2348.



*Supported in part by
a grant from:*

Glaxo

**Allen &
Hanburys**

Division of Glaxo Inc.

**RADIOMETER
COPENHAGEN**

Just-In-Time

Continuing Education for Today's Practitioner

AARC's Professor's Rounds in Respiratory Care

**Live Satellite Videoconferences
That Provide Professional Education
To RCPs In Your Facility**

And You Can Earn Up to Seven Continuing Education Credits.

Part I • The New JCAHO Hospital Standards: Their Effect on Respiratory Care
March 28, 12:30 p.m. to 2 p.m. Eastern Time—Nancy Telford, RRT with Moderator Sam P. Giordano, MBA, RRT

Part II • Prevention and Management of Ventilator-Induced Lung Injury
April 25, 12:30 p.m. to 2 p.m. Eastern Time—David J. Pierson, MD with Moderator Richard D. Branson, RRT

Part III • The Multidisciplinary Team Approach and Respiratory Care
June 20, 12:30 p.m. to 2 p.m. Eastern Time—Kevin L. Shrake, MA, RRT, CHE with Sam P. Giordano, MBA, RRT

Part IV • Emergency Respiratory Care: The Respiratory Care Practitioner's Role
July 19, 12:30 p.m. to 2 p.m. Eastern Time—Charles G. Durbin, Jr., MD, FCCM with Richard D. Branson, RRT

Part V • Noninvasive Mechanical Ventilation: Its Role in Acute and Chronic Ventilatory Failure
August 2, 12:30 p.m. to 2 p.m. Eastern Time—Nicholas S. Hill, MD with Richard D. Branson, RRT

Part VI • Organizing a Respiratory Care Department without Walls
September 19, 12:30 p.m. to 2 p.m. Eastern Time—John R. Walton, MBA, MHA, RRT, CHE with Sam P. Giordano, MBA, RRT

Part VII • Shortening Length of Stay on Ventilators and in Hospitals
November 14, 12:30 p.m. to 2 p.m. Eastern Time—Neil R. MacIntyre, MD with Richard D. Branson, RRT

Registration Fees

	AARC Member	Nonmember
Site Registration		
Single Program	\$260	\$295
All Seven Programs	\$850	\$910
Four Clinically		
Focused Programs	\$635	\$685
Three Respiratory Care Issues Programs	\$485	\$550



A VHA Satellite Network Production

Contact VHA Satellite Network,

Attn: Registration SATNET 4, P.O. Box 140909, Irving, TX 75014-0909, or call (214) 830-0061

A Continuing Education Program of the American Association for Respiratory Care • Produced by VHA Satellite Network

1995 Annual Convention

of the American Association for Respiratory Care
Orlando, Florida • December 2-5, 1995

By Tony Dal Nogare, MD

ARDS was originally described in 1967 and has since undergone much scrutiny. This review of ARDS discusses the latest developments in risk factors and treatment. Also covers the 5 diagnostic criteria that must be present to make an accurate diagnosis of ARDS, including clinical, radiographic, and physiologic criteria.

Item VT31

VHS (60 minutes)
\$35 (nonmember \$40).
Add \$3.25 for shipping.

Call (214) 243-2272 or
Fax your order to
(214) 484-6010

American Association for
Respiratory Care
11030 Ables Ln.
Dallas, TX 75229-4593

Prices subject to change without notice.

**WITH THE
INFORMATION
SERVICE CARD,
YOU CAN STOP
SEARCHING AND
START BUYING.**

Get the facts
on all the
products and
services
advertised in
this issue
easily and quickly.
The computerized
Information
Service Card
does it all.
Simply fill in your
name and
address, check
the appropriate
boxes, and mail
or fax it.

ARDS

Authors in This Issue

Alagar, Ravikumar	498	Hess, Dean R.....	498
Branson, Richard D.....	479	Johannigman, Jay A.....	479
Boudin, Karen M.....	550	Kaye, William.....	538
Fluck, Robert R.....	567	Malinowski, Cynthia.....	575
Hamill, Robin.....	515	Rubinfeld, Gordon D.....	528

Advertisers in This Issue

Armstrong Medical	469	Nellcor	Cover 2
DHD Diemolding Healthcare Div	458	Newport Medical.....	473
Dräger Critical Care	467	Puritan-Bennett Corp	Cover 3
HealthScan Products Inc	465	Respironics	463, 471
Impact Instrumentation.....	460	Sherwood Medical	Cover 4

**For faster
service, FAX
your reader
service card to
(609) 786-4415**

RESPIRATORY CARE

May Reader Service

Expires August 31, 1995

Name _____ Ph# _____
Institution _____
Street _____
City/State/Zip _____

Please Circle No More Than 15 Items

80	81	82	83	84	85	86	87	88	89	90	91	92	93	94
95	96	97	98	99	100	101	102	103	104	105	106	107	108	109
110	111	112	113	114	115	116	117	118	119	120	121	122	123	124
125	126	127	128	129	130	131	132	133	134	135	136	137	138	139
140	141	142	143	144	145	146	147	148	149	150	151	152	153	154
155	156	157	158	159	160	161	162	163	164	165	166	167	168	169
170	171	172	173	174	175	176	177	178	179	180	181	182	183	184
185	186	187	188	189	190	191	192	193	194	195	196	197	198	199

- 81 AARC**
Membership
Information
- 82 RESPIRATORY CARE**
Subscription
Information
- 98 Armstrong Medical**
Video Adapter
- 111 DHD**
MDI Spacer
- 101 Dräger Critical Care**
Vent Monitor
- 123 HealthScan Products**
Peak Flow Monitor
- 118 HealthScan Products**
Optihaler
- 115 Impact Instrumentation**
Portable Ventilator
- 109 Nellcor**
Capnography
- 151 Newport Medical**
VM200 Ventilator
- 102 Puritan Bennett**
7200 Series Ventilator
- 100 Respironics**
Disposable Resuscitator
- 129 Respironics**
REMSTAR/CPAP
- 157 Sherwood Medical**
Ventilation Monitor

RESPIRATORY CARE

May Reader Service

Expires August 31, 1995

Name _____ Ph# _____
Institution _____
Street _____
City/State/Zip _____

Please Circle No More Than 15 Items

80	81	82	83	84	85	86	87	88	89	90	91	92	93	94
95	96	97	98	99	100	101	102	103	104	105	106	107	108	109
110	111	112	113	114	115	116	117	118	119	120	121	122	123	124
125	126	127	128	129	130	131	132	133	134	135	136	137	138	139
140	141	142	143	144	145	146	147	148	149	150	151	152	153	154
155	156	157	158	159	160	161	162	163	164	165	166	167	168	169
170	171	172	173	174	175	176	177	178	179	180	181	182	183	184
185	186	187	188	189	190	191	192	193	194	195	196	197	198	199

- I. Type of Instr/Practice
 - 1 ☐ Hosp ≥ 500 or more beds
 - 2 ☐ Hosp 300 to 499 beds
 - 3 ☐ Hosp 200 to 299 beds
 - 4 ☐ Hosp 100 to 199 beds
 - 5 ☐ Hosp <100 or less bed
 - 6 ☐ Skilled Nursing Facility
 - 7 ☐ Home Care Practice
 - 8 ☐ School
- II. Department
 - A ☐ Respiratory Therapy
 - B ☐ Cardiopulmonary
 - C ☐ Anesthesia Service
 - D ☐ Emergency Dept.
- III. Specialty
 - 1 ☐ Clinical Practice
 - 2 ☐ Perinatal/Pediatrics
 - 3 ☐ Critical Care
 - 4 ☐ Clinical Research
 - 5 ☐ Pulmonary Function Lab
 - 6 ☐ Home Care/Rehab
 - 7 ☐ Education
 - 8 ☐ Management
- IV. Position
 - A ☐ Dept Head
 - B ☐ Chief Therapist
 - C ☐ Supervisor
 - D ☐ Staff Technician
 - E ☐ Staff Therapist
 - F ☐ Educator
 - G ☐ Medical Director
 - H ☐ Anesthesiologist
 - I ☐ Pulmonologist
 - J ☐ Other MD
 - K ☐ Nurse
- V. Are you a member of the AARC?
 - 1. ☐ Yes 2 ☐ No

- I. Type of Instr/Practice
 - 1 ☐ Hosp ≥ 500 or more beds
 - 2 ☐ Hosp 300 to 499 beds
 - 3 ☐ Hosp 200 to 299 beds
 - 4 ☐ Hosp 100 to 199 beds
 - 5 ☐ Hosp <100 or less bed
 - 6 ☐ Skilled Nursing Facility
 - 7 ☐ Home Care Practice
 - 8 ☐ School
- II. Department
 - A ☐ Respiratory Therapy
 - B ☐ Cardiopulmonary
 - C ☐ Anesthesia Service
 - D ☐ Emergency Dept.
- III. Specialty
 - 1 ☐ Clinical Practice
 - 2 ☐ Perinatal/Pediatrics
 - 3 ☐ Critical Care
 - 4 ☐ Clinical Research
 - 5 ☐ Pulmonary Function Lab
 - 6 ☐ Home Care/Rehab
 - 7 ☐ Education
 - 8 ☐ Management
- IV. Position
 - A ☐ Dept Head
 - B ☐ Chief Therapist
 - C ☐ Supervisor
 - D ☐ Staff Technician
 - E ☐ Staff Therapist
 - F ☐ Educator
 - G ☐ Medical Director
 - H ☐ Anesthesiologist
 - I ☐ Pulmonologist
 - J ☐ Other MD
 - K ☐ Nurse
- V. Are you a member of the AARC?
 - 1. ☐ Yes 2 ☐ No



**Information Requests or
Change of Address**

Please complete the card below

AARC Membership No. _____

Old Address

Name _____

Street _____

City/State/Zip _____

New Address

Street _____

City/State/Zip _____

Check the boxes
below for information
from the AARC

- ☐ Change of address
- ☐ AARC Membership Info
- ☐ AARC Catalog
- ☐ AARC Position Statement



NO POSTAGE
NECESSARY IF
MAILED IN THE
UNITED STATES



BUSINESS REPLY MAIL

FIRST-CLASS MAIL PERMIT NO. 439 RIVERTON, NJ

POSTAGE WILL BE PAID BY ADDRESSEE

AARC Publications

PO BOX 11605

RIVERTON NJ 08076-7205



NO POSTAGE
NECESSARY IF
MAILED IN THE
UNITED STATES



BUSINESS REPLY MAIL

FIRST-CLASS MAIL PERMIT NO. 439 RIVERTON, NJ

POSTAGE WILL BE PAID BY ADDRESSEE

AARC Publications

PO BOX 11605

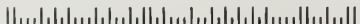
RIVERTON NJ 08076-7205



Place
Stamp
Here

Return Address

AMERICAN ASSOCIATION FOR
RESPIRATORY CARE
11030 ABLES LN
DALLAS TX 75229-4593



Use these
cards to
request
information
from the
AARC or
advertisers
in this
issue.

We'd like to introduce a special new feature of our 7200[®] Series Ventilator: Insight.

The best clinical decisions require insight. And the best insight comes from the best tools. Like the new 7200 Series Ventilatory System.

You've always known the 7200 Series for its reliability and upgradeability. Now we're adding a whole new dimension – five enhancements that allow greater insight into the patient's status.



The rapid shallow breathing index (RR/Vt) is among over 30 trendable parameters in the new Graphics 2.0 option.

Graphics 2.0 allows you to select from a menu of over 30 parameters to be continuously trended for up to 72 hours. With just two simple key

presses, the 7250[™] Metabolic Monitor accurately and continuously monitors whole body O₂ consumption, CO₂ production and calculates energy expenditure for ventilated patients. *Auto-PEEP* provides reliable intrinsic

The 7250 Metabolic Monitor continuously measures and calculates several metabolic parameters including energy expenditure.

Its unique sampling method is designed to improve measurement accuracy on mechanically ventilated patients.

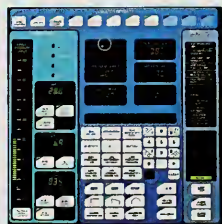


PEEP and circuit PEEP measurements. *DCI 2.0* allows two host devices such as bedside monitors and computers to be linked to the ventilator simultaneously. And finally, the *Enhanced-Plus* keyboard allows direct access to options and shows you at a glance which are enabled.

Together, the 7200 Ventilator and these new upgrades give you a greater ability than ever before to observe patient responses to ventilatory titration.

The new 7200 Series. Now turning information into insight.

For more information about our *New Tools for Greater Insight[™]*, contact your Puritan-Bennett sales representative or call 1-800-255-6773.



Option keys on the new, *Enhanced-Plus* allow direct access to options and identify which are active. The new bar graph provides exceptional visibility and resolution to changes in pressure.

1994 Zenith Award Winner

WE'RE IN IT FOR LIFE[™]

PB PURITAN BENNETT

RESPIRADYNE® II PLUS

PULMONARY FUNCTION/VENTILATION MONITOR Graphic Printouts...Multi-Patient Memory...and Easy to Use

Results-Oriented Features At Cost Effective Prices

- New Graphic Forced Vital Capacity (FVC) document printout of Flow vs Volume and Volume vs Time
- New 10 patient memory with 8 pre-bronchodilator and 8 post-bronchodilator tests per patient and automatic calculation of % change
- New customizing software package
- New Slow Vital Capacity (SVC) monitoring
- Automatic determination of "best test"
- Knudson, ITS and ECCS reference nomograms
- Easy to operate



Performs A Complete Range Of Test Measurements

Forced Exhalation Parameters

- Forced Vital Capacity (FVC)
- Forced Expiratory Volume in One Second (FEV_1)
- FEV_1 /FVC Ratio
- FVC Time
- Peak Flow
- Forced Expiratory Flow Between 25% and 75% of Vital Capacity ($FEF_{25-75\%}$)
- Percent Extrapolated Volume (Vol. _{EXTRA}%)

Weaning/Extubation Parameters

- Respiratory Rate (RR)
- Tidal Volume (TV)
- Minute Volume (MV)
- Slow Vital Capacity (SVC)
- Maximum Voluntary Ventilation (MVV)
- Negative Inspiratory Force (NIF)

For further information, call:

1-800-325-7472 (outside Missouri)

1-800-392-7318 (in Missouri)

Sherwood
MEDICAL
ST LOUIS MO 63103 U.S.A.

Circle 157 on reader service card